GOVERNMENT SIDDHA MEDICAL COLLEGE
PALAYAMKOTTAI, TIRUNELVELI-627002
TAMILNADU, INDIA.
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BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"A Clinical study of KARISALANKANNI CHOORANAM in the management of VATHA PANDU (Iron Deficiency Anemia)"** is a bonafide work done by **Dr.L.ILAMATHI (Reg No: 321311003)** in partial fulfillment of the University rules and regulations for award of **M.D (Siddha), Branch I-** ***Pothu Maruthuvam*** under my guidance and supervision during the academic year **October 2013-2016.**

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SIMAI
Siddha a timeless healing art

Siddha Medical Association of India

Siddha Workshop Certificate

This is to certify that Dr. L. Ilamathi Bearing Reg.No. 3969 Registered with Tamilnadu siddha Medical Council, Address ..... has participated as Faculty / Delegate in Oneday Workshop on topic "Siddha Orthopedics" held on 29th May, 2016 at Gandhi Bhavan, Thiruvananthapuram. Travancore - Cochin Medical Council has Granted Six Credit hours for faculty/ delegates.

SIDDHA MEDICAL ASSOCIATION OF INDIA
Date: 29/05/16
Reg. No. TVM/KL/1063/2015
KERALA

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Secretary
(SIMAI)

Dr.V.B Vijaya Kumar
Elected CCIM Member (Siddha)
Kerala.

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GOVERNMENT SIDDHA MEDICAL COLLEGE
PALAYAMKOTTAI

CME

Conducted by

POST GRADUATE DEPARTMENT OF POTHU MARUTHUVAM

Certificate

This is to certify that Dr. L.. ILAMATHI

of P.G. Pothu Maruthuvam *department has participated in the*

Continue Medical Education Programme on **Renal Diseases**

held at **Government Siddha Medical College Palayamkottai** *On 08 - 06 - 2016 Wednesday*

Prof. Dr. A. MANOHARAN M.D (S)
H.O.D of P.G. Pothumaruthuvam
Government Siddha Medical College, Palayamkottai

Prof. Dr. S. VICTORIA M.D (S)
Principal,
Government Siddha Medical College, Palayamkottai

CERTIFICATE

Certified that I have gone through the dissertation submitted by **DR.L. ILAMATHI(Reg No: 321311003)** a student of final M.D (s) Branch I **Pothu Maruthuvam** of this college and the dissertation work has been carried out by the individual only. This dissertation does not represent or reproduce the dissertation submitted and approved earlier.

Place : Palayamkottai

Date:

Head of the Department

P.G Pothu Maruthuvam

Govt. Siddha Medical, Palayamkottai

GOVERNMENT SIDDHA MEDICAL COLLEGE

PALAYAMKOTTAI

Certificate of Botanical Authenticity

Certified the following plant drugs used in Siddha formulation Karisalankanni Chooranam (Internal) for the management of VathaPaandu (Anaemia) taken up for Post Graduation Dissertation Studies by Dr.L.Ilamathi (Reg No.321311003) PG Dept, of Pothu Maruthuvam are correctly identified and authenticated through Visual inspection / Organoleptic Characters / Experience, Education & Training Morphology Microscopical and Taxonomical methods.

S.N	Name	Botanical Name	Parts used	Quantity
1.	Karisalankanni	*Eclipta prostrata*	Dried Leaves	4 parts
2.	Mookiratai	*Boerhaevia diffusa*	Dried Whole plant	1 part
3.	Chukku	*Zingiber officinale*	Dried Rhizome	1 part
4.	Milagu	*Piper nigrum*	Dried Seed	1 part
5.	Thippili	*Piper longum*	Dried Fruit	1 part
6.	Kadukkai	*Terminalia chebula*	Dried Thol	1 part
7.	Nellikkai	*Phyllanthus emblica*	Dried Fruit	1 part
8.	Thandrikai	*Terminalia bellerica*	Dried Fruit	1 part
9.	Maramanjal	*Coscinium fenestratum*	Dried Wood	1 part
10.	Thaniya	*Coriandrum sativum*	Dried Fruit	1 part
11.	Athimathuram	*Glycyrrhiza glabra*	Dried Root	1 part
12.	Karunseeragam	*Nigella sativa*	Dried Seed	1 part
13.	Thalisapathiri	*Abies spectabilis*	Dried Leaves	1 part
14.	Elam	*Elettaria cardamomum*	Dried Seed	1 part
15.	Seeragam	*Cuminum cyminum*	Dried Seed	1 part

Station: Palayamkottai

Date: 21.9.2015

Authorized Signature 21/9/15

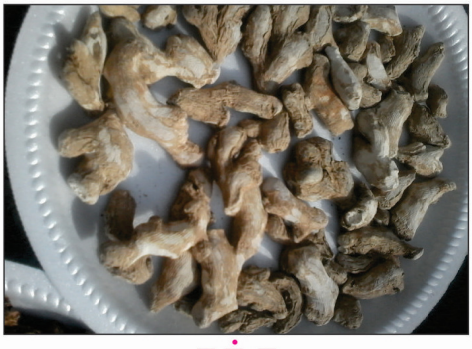
Dr. S. SUTHA, M.Sc.,M.Ed.,Ph.D.,
Associate Professor
Dept. of Medicinal Botany
Govt. Siddha Medical College
Palayamkottai, Tirunelveli - 2.

கரிசலாங்கன்னி சூரணம் சேரும் பொருட்கள்

கரிசலாங்கன்னி

மூக்கிரட்டை

நெல்லி வற்றல்



சுக்கு

மிளகு

ஏலரிசி

தான்றிக்காய்

தனியா

கரிசலாங்கன்னி சூரணம் சேரும் பொருட்கள்

அதிமதுரம்

கருஞ்சீரகம்

மரமஞ்சள்

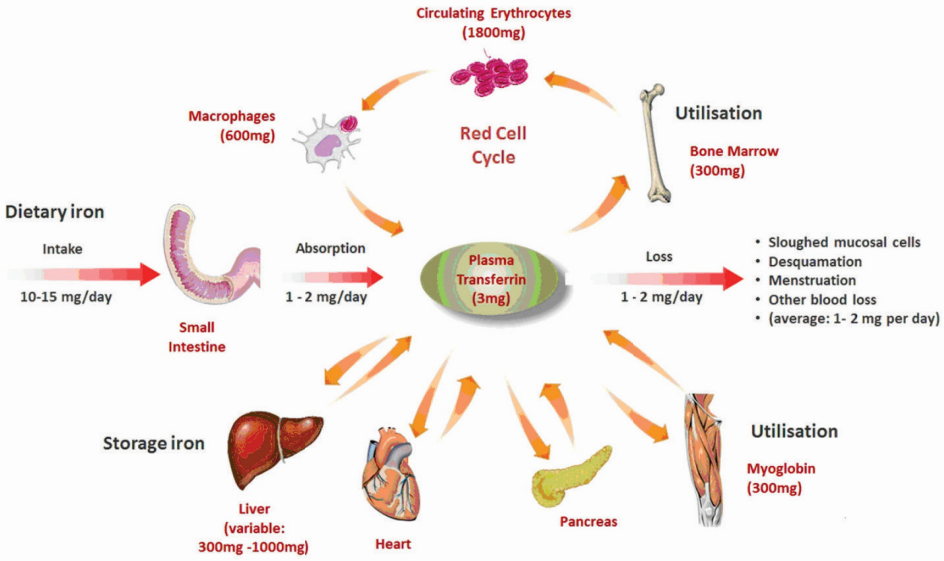
சீரகம்

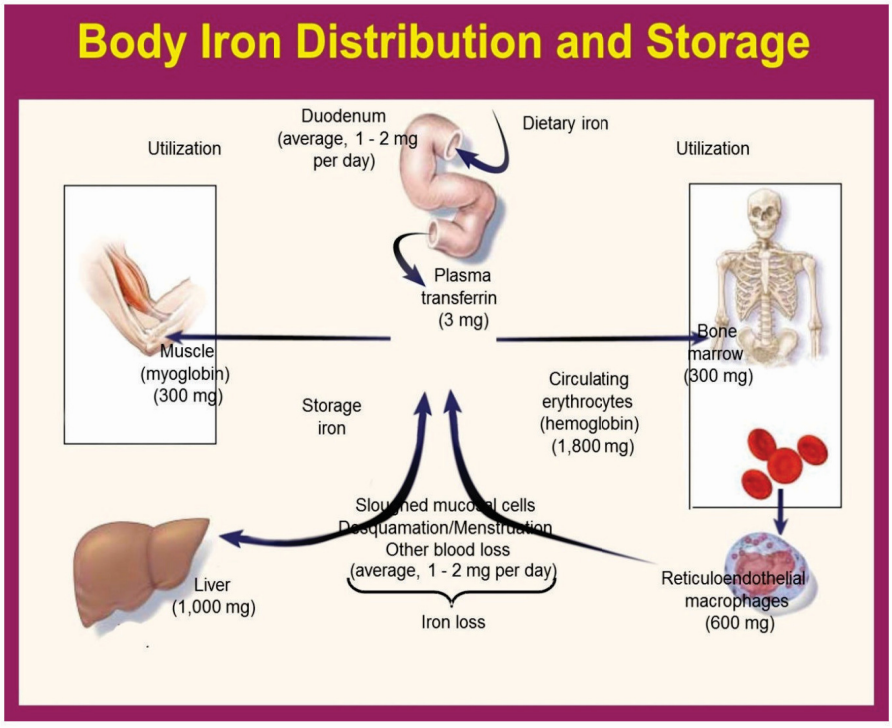
திப்பிலி

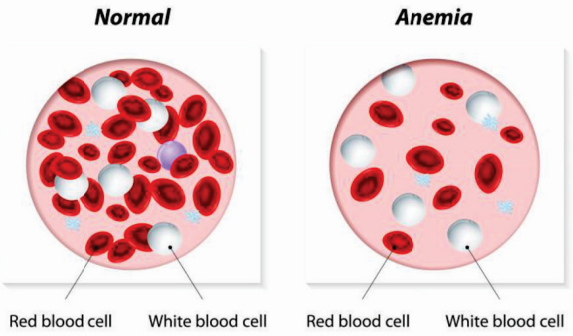
தாளிசபத்திரி

கடுக்காய் தோல்

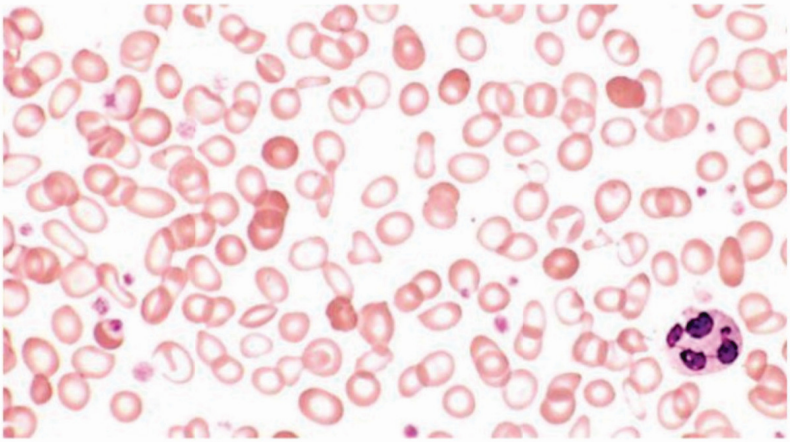
IRON METABOLISM



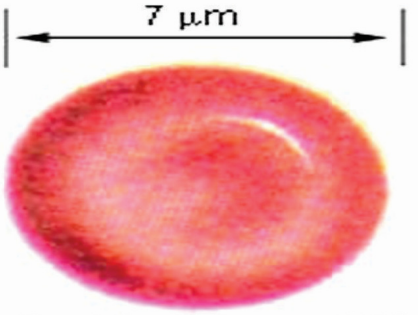




Microcytic hypochromic anemia of iron deficiency (peripheral blood smear)



Structure of RBC



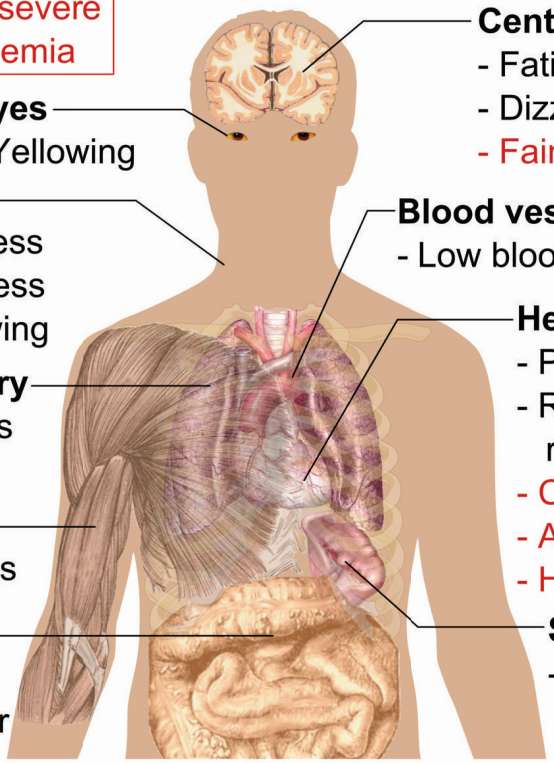
Top View shows RBC to be circular

Side view shows RBC

Symptoms of

Anemia

Red = In severe anemia



Central
- Fatigue
- Dizziness
- Fainting

Eyes
- Yellowing

Skin
- Paleness
- Coldness
- Yellowing

Blood vessels
- Low blood pressure

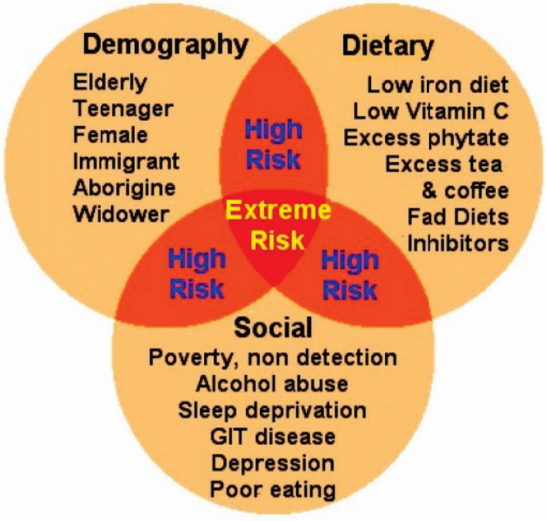
Heart
- Palpitations
- Rapid heart rate
- Chest pain
- Angina
- Heart attack

Respiratory
- Shortness of breath

Muscular
- Weakness

Intestinal
- Changed stool color

Spleen
- Enlarge-ment



கரிசலாங்கண்ணி சூரணம்

Introduction:

Siddha system of medicine is the **divine medicine,** which was founded by siddhars, it is considered to be one of the ancient traditional medicine in the world, Siddha system of medicine is prevalant before tholkappiam it was proved by some of the literary evidence.

Panchabootham and uyir vayus are responsible for the organsiation of this body and universe from the microcosam and macrocosam. When the equilibrium of this panchabootham and uyir vayus are distrubed various disease are formed in our body.

Medicine is not merely a science but an art as well. This science is fundamently important to man's well being and survival.

The present concept of poverty and malnutrition is a major cause for disease. In siddha three vital humours are major role to maintain the body. The deranged humours (or) Altered humours can produce the diseses. In this view thiruvalluvar in thirukural says,

"ÌÂ‹ °¬øJÂ‹ «ï£Œ ªêŒ»‹ Ë«ô£˜

õO º îô£ â‡ E ò Í ¡Á" ÜFè£ó‹&941

Derangements of these three thathus causes disease. In developing countries like India "pandu noi" is more prevalant.

The symptoms of the disease can similarly correlate in Modern science as Iron defeciency anaemia. The word aneamia actually means "Lack of blood" in greek. This disease affects 1 in 5 people in the developed world. It affects the person who suffer from malnutrition and chronic diseases.

According to siddha concept, it is caused due to disturbances in normal blood. Which is one of the seven humour of the body (Seven UdalThathukkal).

"**Yugimuni**" the great siddhar quotes seven humour as follows.

Yugimunivar clearly mentioned that blood plays an vital role in forming seven humours.

"ªê¡¬ñ ñ£˜„ gó‰ ªî£‡ È ŸÁøƒ° ôñ£‹

ªêŒA¡ø õóõ˜èœ ¬è£«ôî£¡

î¡ùñ£‹ óê ñóˆîñ£ ƒèêº«ñ¬î

î¬ê ñ„¬êªò£´ ²,ô‰î£ «î£ö£A

à‡ñ£ º¼ ªõ‡ µ ëêgóñ£A

à J˜ ê†¬ì óê...«ê˜ˆ¶ «ó£ñ£A

õ¡ùñ£‹ õ£î HˆîF «ê†ðñ£A

õ¼A¡ø ê†ìˆFQì õ‡¬ñ «è«÷."

--Î A ¬õˆFò C‰î£ñ E

The seven humour are formed from panchaboothas by pancheekaranam.This concept was quote by "**Thirumoolar in Thirumandiram** as,

"Þóî‹ à Fó‹ Þ¬ø„C «î£™ «ñ¬î

ñ¼Mò õˆF õ£°‹ ªð£´ñ„¬ê

ðóMò ²,Aô‹ ð£ö£‹ à ð£F

à¼õñô£½ì¡ å¡ªøùô£«ñ" -- F¼ñ‰Fó‹ -2086

Vathapandu affects many crores of people in the world . About 20% of world population is prevalent to Anaemia. Environmental pollution,Lack of well balanced diet , infections and poisonous substance are the promotors of this disease.

Though this disease can be diagonsed and treated easily.If untreated in advance stage it may affects important vital organs like Heart, kidney, digestive system. Untreated pandu noi may leads to sobai (oedema) and kamalai(Jaundice)

So, I have selected herbal formulation "**Karisalankanni chooranam**" to asses clinical and therapeutic effect in the management of **Vatha Pandu noi**.

Both the good diet and the better absorption brings the best efficacy.This drug contains **Karisalankanni** which is rich in Iron,mookirattai which acts as an Liver tonic, Kadukkai, Nellikai, Thandrikai which is known as "Triphala" is added which helps for the absorption of iron and various other drugs are added are more efficient in curing this disease. This work present with prestige about the various clinical findings clinical assessment,various studies in the treatement of vathapandu.

AIMS AND OBJECTIVES

The Aim of this works is to make an observation of aetiology, pathogenesis, clincial features, treatment, prognosis of vatha pandu noi.

For the purpose of diagnosis the eight types of clinical investigation of siddha medicine (Ennvagai thervugal) and relavant modern diagnostic methods are also to be adopted.

The selection of cases is to be made from the OPD-Department of Pothu Maruthuvam , Government Siddha Medical College and hospitals, Palayamkottai.

The patients are given with "**Karaisalankanni chooranam**".

The dynamic approach deals very much in detail with the work done with devotion and dedication to bring out the following objectives are,

1. To collect various siddha literatures dealing with nomenclature, defintion, aetiology,classification, signs and symptoms, Naadi, diagnosis, diet, prognosis, complications of "Vatha Pandu Noi" in Siddha system of medicine.

2. To know the extent to which the correlation of Aetiology,classification, signs and symptoms of Pandu Noi in Siddha medicine as well as modern medicine.

3. To expose the efficacy of Siddhar's diagnostic principles. (Envagai Thervugal)

4. To have some idea about the disease like incidence, prevalance and surveilance of the disease.

5. To know the clinical and therapeutic efficacy of the trial medicine.

6. Biochemical, pharmocological studies which have been ruled out to find the potency and free toxic effect of the trial drug.

7. To evaluate the safety profile drug of the trial medicine Karisalankanni Chooranam.

REVIEW OF LITERATURE

Siddha Literature

Siddha system is said to have orginated from lord siva. Agasthiyar was the first disciple of lord siva. He was the pioneer to propogate this siddha system of medicine.

The siddha system of medicine is based on the tridhosa theory. According to which the human system is mediated by three vitiatling elements (or) thathus which is the functional units of living beings namely vatham, pitham and kabam.

Diseases are produced by disequilibrium of this three vitaling elements which may be due to various causes like dietetic regimen, life style pattern and ecological imbalance etc..

The diseases “Vatha Pandu” has its historical importances. The word “Pandu” has been derived from the great hindu epic “MAHABHARATHAM” where the father of five heros “PANCHA PANDAVAR” is “Pandu”. It is said this man when born was very pale and anaemic and this condition was named after him as “Pandu”.

SYNONYMS OF PANDU

Vemai Noi, velluppu Noi, ven pandam.

IYAL (Definition):

Pandu is the diseases of raktha thathu charactertics by pallor of skin,nail beds and conjuction. The great siddhar **Agasthiyar** define pandu as,

“²Nõ£°‰î «îèñŠð£ è£ í ˆî¶ õ‰î£Œ

õŸPM´ ñ¡ùõ£ê™ «è†A™

ðNè£ó˜ ºèˆFQ™ ºNò£˜ «ð£«ô ð£‡ìªñô£‹

ªõÂ,è®ˆî¶ óˆî‹”

&Üèˆfò˜¬õˆfò è£Mò‹.

“«îèˆF™ Þóˆî‹ õŸP

béè£ù Mˆî «ï£Œ è£µ ñŠð£”

-&Üèˆfò˜° í õ£èì‹.

The blood is subjected to various researches from ancient days. It was also believed that the man’s life is decided by blood in those days. Blood acts as mirror that reflects any pathological changes in the body.

According to **pattinathar** these are crores of uyir anukkal in blood. He says,

“«ñMò ¹¡ñJ˜ˆ ªî£¬è«ò Ü‹ñJ˜

ð£Mò «î£L¡ ðóŠ«ð£ «î£Q¬ìŠ

¹èL†´Š ªð£Fˆî ¹‡«í£ ¹‡ E¬ì

áÁ‹ àFóŠ ¹ù«ô£ ÃÁªêŒ¶

Þ¬ìJ¬ì GŸ°‹ â½‹«ð£ â½‹H¬ì

º¬ìªè¿ Í¬÷ M¿«î£ õ¿õ¿ˆ¶

à œO ¬ ì ª ò£ º ° ‹ õ ¿ ‹ «ð£ ª ñ™ô G ¡ Á

á Á ‹ ¹ ¿ M ¡ å ¿ é «è£ cK ¬ ì …”

& ð†® ù ˆ î£˜

(F¼M ¬ ì ñ¼ É ˜ º ‹ ñ E , «è£ ¬ õ)

According to siddhars. As the digestion takes place in the body Rasathathu is formed on the very first day. On the second day Raktham is formed it is followed by the formation of mamisam,kozhuppu,enbu,moolai, sukkilam or suronitham on respective days that is on 3rd, 4th, 5th, 6th and 7th days respectively.This is called as udalthathukal.It is to be noted that the nutrients absorbed after digestion are responsible for the metabolic functions and the formution of blood.

NOI VARUM VAZHI:(Aetiology)

According to "YUGI MUNIVAR ". in yugivaithiya chinthamani clearly mentioned the etiopathogenises of the diseases.

“ Ü P‰ ¶ «ñ à Ÿð ˆ F ª ê£™ô , «è ÷ £Œ

Ü F ê£ó ñ ÷ I ÷ A ª ò‰ « ï ó‰ î £ ¡

HP‰ ¶ «ñ ¹ O » Š¹ ª ð¼ ˆ î £½ ‹

ª ð ˆ î ñ£ ñ , A Q J L¼‰ î ô £½ ‹

I P‰ ¶ «ñ î £ ‹ Ì ô I è Ü¼‰ î ô £½ ‹

eP « ò ñ ¶ , è ¬ ÷ ^ î £ ¡ ¹ C ˆ î £½ ‹

ðP‰ ¶ «ñ ðè™ G ˆ F ¬ ó « ò ª êŒ î ô £½ ‹

ð£ ‡ ´ õ‰ ¶ ð£ K½œ « ÷ £˜ ð ´ ‹ ð£ ì £ « ñ .

ð£ì£ù ð...² î ¬ ù F¼® «ù£˜, ° ‹

ð£ƒè£ù Cõ ¶J ¬ ô î^ F¼® «ù£˜, ° ‹

ñ£ì£ù ð² õ ¬ îŠ ð†®Q ò£è

¬ õ, A ¡ «ø£˜ ñ ¬ øõNJ™ ï ì‰Fð£˜

è£ì£ù õ£ó E ò^ îQŸ ðP^¶,

è´ õ ¬ îèœ ªêŒA ¡ «ø£˜èœ è ‡ è£ í £î

«è£ì£ù ðNªê£™L, °®ªè¼, ° ‹

ªè£´ ‹ ð£M ð£ ‡ ´ Mù£Ÿ °F ªè£œõ£«ó”

& ÎA ¬ õ^Fò C‰î£ñ E

Yugi muni says that wrong life style excessive intake of food stuffs with salt and sour taste, staying for long time in hot climate. excessive intake of pan and alchol , sleep in the daytime leads in pandu.

2. In Agathiyar vaidhyam - 80

Pandu is considered as one among the kanma noi.

“° J™õ£Œ °wì‹ êòƒ° ¡ ñ cKN¾ ²óƒAó£E

có ¬ ìŠ¹ ð£ ‡ ´ Íôõ£Œ¾

è ¡ ñõ£» õ¼ƒè ‡ E™ °‰î£Œ è®ù‰ îêõ£Œ¾

è£ í õ£è º ¡ ªêŒî à J˜èÀ ‹ M ¬ ù î£«ù”.

In Agasthiar paripooram-400.

“ªê™ô£î è®Mìƒèœ ° ¡ ñ ‹ ð£ ‡ ´

¶ò˜ bó£, è ¡ ñM ¬ ù ªêŒî ð£õ ‹

â™«ô£¼‹ ï¬è‚è à´‹ªð´ˆî ð£M

Þ¡ù º ‡ ´ Mðóñ£Œ à ôAŸ«è«÷”.

è®Mì ƒ èœ - Hemolysis due to toxins

°¡ñ‹ - Acid peptic disease

è¡ñM¬ ù-Abnormal morphological changes in RBC(Genetic Disorder).

In **Agasthiyar Gunavagavagadam**:

“ªè£œ÷ì£ Üð‚°õ «ð£êùˆFù£½‹

°®ªè´ˆî ªð¼‹ð£´ Aó£ E ò£½‹

èœ÷ì£ è¼Šðˆ F¡ Aó£ E ò£½‹

èùñ£ù óˆîˆF¡ «ð£‚Aù£½‹

Üœ÷ì£ ÜFò£ñ èõùˆî£½‹

Ü÷õŸø Mê£ó‰î£ù¬ì»‹ «ð£¶‹

ªîœ÷«õ «îèˆF™ óˆî‹ ªè†®

ªîOõ£ù ð£‡ì¶ ¾ º ‡ì£‹ ð£«ó”.

Mal nutrition

Dysfunctional utrine bleeding

Ulcerative colitis

Bleeding disorders.

According to agathiyar Improperly cooked food, food habits, Imbalanced diet, menorrhagia in females, chronic Diarrhoea, karupa kirani, blood loss due to various aetiology, stress and strains are the causes of pandu.

In Thanvanthiri vaidhyam:

“F¼‰F´‹ ð£‡´ «ó£è… «ê˜‰F´‹ °íˆ¬î, «è÷£Œ

Þ¼‰F´‹ õ£î HˆîC«ôŸðù I¬õî£¡ ñ£Á‹

ðK‰¶ î£ªù£¡«ø£ ªì£¡Á ªð£¼‰¶õî£½ ñ‡«í£

ì¼‰¶õî£½‹ ð£‡´ Ü¬í‰F´ ªñ¡ùô£«ñ

ÝAò Íô‰î¡Q ô¬í‰î¾†ìíˆFù£½‰

«îè «ð£û¬í »œ÷£˜,°ˆ îKˆFó… «ê˜îô£½‹

«õèñ£‰ FK«î£-ûéèœ IëC«ò ð£‡´õ£«ñ.”

Imbalance between three thathu, excessive heat, pica (eating of sand), accumulation due to abana, sorrow, psychological factors may lead to vitation of three thathus and cause pandu.

Due to worm Infestations:

“õò™îQ«ô Ìï£è ñ‡¬íˆ î£«ù

õ¼‰Fò¶ ¹ˆ¶ «ð£ô õ‰¬îò£°‹

ðJ™ ªñ£Nf˜ «îèˆF™ A¼Iî£«ù”

& °¼ï£®

Pathological loss of blood may occur due to various cause one among them is worm infestation which leads to Iron deficiency anaemia.

In **Agasthiyar Gunavagadam**:

“ï£ªñ£¡Á ªê£™½A«ø£‹ ð£‡´ õ‰î

ïôñ£ù è¼ññ¶ ªê£™ô, «èÀ

î£ªñ¡ø î£Œî‰¬î ñù‹«ï£è ªêŒî™

îó E îQ ½œ÷õ˜, ° ‹ Ü ¡ «ð ªê£™ô

ô£ªñ ¡ø M¼ŠðNˆî™ ªêM ²õ£ê

ñŸÁ‹ ªð£Œªê£™ô£ƒ è£ó...ªêŒî™

«õªñ ¡ø è¼ññ¶ Hˆî «ñP

ªõÁˆîîì£ ªõŠ¹ I...C, ¬èè£ô«ô£Œ”

Agasthiyar say that Irresponsiblity and disobediance to parents, speaking lie, getting anger on other’s stimulates pitha dhosam which results in pandu Noi.

In **Therayar mahakarisal**:

“õ£î ð£‡´ «ï£«ò î£Â‹ õ‰F´‹ ðKê«è÷£Œ

ê£î‹ cg„¬ê »ŸÁˆ î¡Qøé è¼F õ£®„

Å¶«ê˜ ªê£¼ð º‡ì£Œ„ ²«ó£E î‹ õŸøõŸø

Ýîóõ£ñ¬ùˆ¶‹ ÜFêò ñ£¼ƒ è‡ìñ£Œ”

“ **Theryar vagadam**” Denotes causative factor of pandu as

“è¼Fò eQ¡ ºœÀ‹ èôˆ¶I ªïŒJ¡

ñ¼Mò â½‹¹ƒ è™½‹ ñƒ¬èò˜ ñ¼‰F´‹

ð¼Aò ðö...«ê£Ÿø£½‹ ðöñô Gø¬èò£½‹

ñ¼Aò ñJ˜è÷£½‹ õ‰F´‹ «ï£JîP«ñ

¹øõ¬ó »‡¬è ò£½‹ «ð£è c¼‡¬èò£½‹

²¼÷«õ ºì,A, ªè£‡´ ºø,Aì, ¬èò£½‹

ñ¼÷«õ «ñ´î¡Q™ ñùºø, Aì,¬èò£½‹

ªð¼è ªõ‡ð£®JJù£½‹ Hø,è «ï£ªò¡Áè£« í ”

Intake of fish bones, paddy brane, small and store old rice, hairs which can produce pandu secondly severe constipation, polluted water, sleeping in abnormal posture causes veluppu Noi.

Noi enn (Classification):

According to **yugimunivar** Pandu Noi is classified into 5 types. This was seen in his quote.

"Ãø«õ ð£‡´MìŠ ªðò¬ó, «è÷£Œ
°PŠð£è ¬õ‰¶ Mîñ£°‹ ð£¼
õ£ó«õ õ£îñ£‹ ð£‡® «ù£´
ñ£˜,èñ£‹ HˆîˆF¡ ð£‡´ î£Â‹
«îø«õ C«ô†´ññ£‹ ð£‡´ î£Â‹
FK«î£ìŠ ð£‡«ì£´ Mìð£‡ ì£°‹"

Vathapandu

Pithapandu

Kaphapandu

Mukkutra pandu

Vishapandu

are 5 types of pandu Noi according to yugi muni.

Thanvanthiri classification:

Thanvathiri classifies pandu Noi into 7 types they are,

"ðJˆFò ð£‡´ õ£î ð£‡´«õ C«ôˆ¶ñ ð£‡´
MòˆFK«î£û ð£‡´ ªõÀ‹ HˆîC«ôˆ¶õ ð£‡´

ðJˆFò õ£î ð£‡´ ðè˜ê‰G õ£î ð£‡´

ïòŠ¹Á‹ ð£‡´ «õN¡ °íˆ¬îï£¡ïM½ ½Ÿ«ø¡”

Vatha pandu

Pitha pandu

Kapapandu

Mukkuta pandu

Pithavatha pandu

Pithakapha pandu

Sannipatha pandu

Agasthiyar classification:

Pandu Noi was classified into 5 types by agasthiyar

“ð£óì£ ð£‡´õ¬è ªê£™ô, «è÷£Œ

ðKõ£ù ð£‡ì¶ î£ùë «êò£°‹

õ£óì£ õ£îHˆî‹ Yî ð£‡´

õ¬èò£ù Mìð£‡´ Iˆ¼Fè£ ð£‡´”

& Üèˆ Fò˜ °íõ£èì‹

Vatha pandu

Pitha pandu

Kapha pandu

Visha pandu

Miruthika pandu

Classification of pandu in various siddha literature are given below.

In **Jeeva Rakshamirtham**:

Vathapandu

Pithapandu

Kapha pandu

Mukkutra pandu

Nanju pandu

Mirthikapuktha pandu

Alimuga pandu

In **Sigitcharatna deepam**:

Vatha pandu,Pitha pandu, kapha pandu, thiridhosa pandu, vishapandu

T.V Sambasivam pillai dictionary

Vathapandu

Pithapandu

Kapha pandu

Mukkutra pandu

Oothu pandu

Neer pandu

Eri pandu

Visha pandu

Roga nirnaya saram:

vathapandu

pithapandu

Kapha pandu

Mukkutra pandu

Visha pandu

Anubava vaidhya devaragasiyam:

Vathapandu

Pithapandu

Kapha pandu

Mukkutra pandu

Nanju pandu

Mirthika puktha pandu

Most of the siddha litrature clearly mentioned the disease based upon kutrangal.

Noi Kurikunangal (clinical features):

Patients experiences the symptoms like fatigueness, difficult in breathing on exertion, blurred vision, giddiness, palpitation, pallor of skin,nail beds, conjuctiva, headache,anorexia.

According to yugi munivar:

“ªè£œ÷«õ õ£î ð£‡´ «ó£èé«è÷£Œ

°ì™Hó†® ò®õJÁ î£¡õL,°‹

îœ÷«õ î£èªñ£´ ðC»I™¬ô

îöô£ù ²ó²óŠð£èˆ î£Â‹

ïœ÷«õ ïó‹ªð™ô£‹ èÁŠ¹ñ£°‹

ï ´ ,èªô£´ è ‡ CõŠ¹ ñôð‰î‰î£¡

M ‡ ÷«õ î¬ôõL , ° ‹«ñQ i *f* ° ‹

ªõ ÀŠð£° ‹ õ£îˆF¡ ð£‡ ´õ«ñ".

Abdominal discomfort, decreased appetite and intake of water, dryness of skin, tremor redness of eyes, constipation, headache, generalised oedema, pallor of skin are the clinical features of "vatha pandu".

According to agasthiyar gunavagadam:

"à ‡ ì£° ‹ «õ¬÷ î¡Q™ «îè «ï˜¬ñ

à ÁFò£Œ„ ªê£™½A«ø¡ ï¡ø£Œ ð£¼

° ‡ ì£ù ºè‹ è ‡ èœ à î´ ï£,°

° PŠð£ù õ£Œ«õ° ‹ «îèºŸÁ‹

ªõ ‡ ì£è «õ»ô˜‰¶ ªõ ‡ ¬ñò£A

Mó™ ïè*f* èœ º¿õF½‹ óˆî‹ õŸP

è ‡ ì£ è£™èœ î£¡ î E‰¶ GŸ° ‹

è¼õ£ù ï£®ò¶ ªñ¶õ£Œ «ð£«ñ" (23)

"«ð£«ñî£¡ bðù *f* èœ ñ†´Šð†´

ªð£Lõ£ù è ‡ MNèœ ªð¼ˆ¶ˆ «î£¡Á‹

Ý«ñî£¡ ÜêF»ñ£ò£ê *f* è ‡ ´

Üõ˜ ï¬ì»‹ î÷˜‰¶ ªð¼Í „²,è ‡ ´

Í«ñî£¡ Í˜„¬ê»ì¡ ñ£˜ ¶®ˆ¶

º®õ£ù è µ ,è£L™ i ,èº ‡ ì£Œ

É «ñ î£Q¼î ò ˆ F ¡ õ î ù‰ î ¡ QŸ

¶¼ˆ F Gè˜ ê ˆ î ñ ¶ «è† ° ‹ ð£«ó” (24)

& Üè ˆ F ò˜ ° í õ£è ì ‹.

Pallor of skin, conjuctiva, tongue, lips and nail beds, dryness of skin, loss of appetite triedness, giddiness, breathlessness on exertion, palpitation,fatigueness, pedel oedema, Increase heart rate are the main symptoms of this disease.

Thanvanthiri vaithiyam:

“ Ý ù è ‡ ñ ô ê ô ƒ è œ Ü E ï è ƒ è Á Š ð î £ ° ‹

î £è ñ£ ñ ƒ è ª ñ ƒ ° ‰ î î ®ò® ð ´ ¬ è « ð£« ô

e ù ñ£Œ ï ´ , è º ‡ ì £ I ¬ ê‰ î ¶ ˜ ð ô º º ‡ ì £

ñ£ù c˜ õ£ î ð£ ‡ ´ ª õ ¡ ù õ ° , è ô£« ñ”

& î ¡ õ‰ FK ¬ õ ˆ F ò ‹ 500

Black colouration of eyes, faeces, nails,urine, pain all over the body, tremor, fatigueness, are the symptoms of vathapandu Noi.

Anubava vaidhya deva ragasiyam:

Discolouration of nail, eye, tremor, generalised body pain, dropsy, fatigueness dryness of skin,dullness of face, abdominal pain,loss of apetite and thirst.

Jeeva rakshamirtham:

Vatha pandu is characterised by generalised body pain,nail beds,faeces and urine are dark in colour constipation,headache, dull pain over chest and abdomen ,loss of apetite.

Vaidhya sara sangiraham:

Swelling and pain over the lower extrimities pallor of skin, lips, eyes,tongue ,urine, dryness of skin, flatulent of abdomen are the feature of vatha pandu.

Roga nirnaya saram:

Pain all over the body,tremor, discoloration of nail, conjuctiva, headache dryness of skin are the symptoms of vatha pandu.

Mukkutra verupadugal (Patholgy of vatha pandu):

Our siddha is based upon thirdhosic theory due to extrinsic and intrinsic factor the pitham in the body gets altered and the digestion is affected.In the blood stream the ranjagam which is the branch of pitha which gives colour to the blood is unable to entertain these ill digested and improperly assimilated nutrients with raktha thathu these defect manifest in the haemopoisis and produce anaemia with the symptoms of pallor of tongue, conjuctiva, nail beds and skin.

The detoriation of kapha thathu with pitham and vatham produces oedema these altered dhosha's finaly alter the normal structure and function of seven thathus which are called Udal thathukal.

Piniyari muraimai (Diagnosis)

Piniyari muraimai is the method of diagonsis the disease affecting the man It is based upon three main principles they are.

Poriyal arithal - Physical examination, Perception

Pulanal arithal - On examination

Vinathal - Symptoms and Signs

Pori is considered as the five sense of perception namely nose, tongue, eye, skin, and ear. While pulan are five objects of senses they are smell, taste, sight, sensation and sound. Vinathal is obtaining information regarding the history of the disease, its clinical features etc... From the patient or his immediate relatives who are taking care of him when the patient is a position to speak or the patient is a child.

The above principles corresponds to the methodology inspection, palpation and interrogation of modern medicine.

Alavai is a parameter through which one can assess the properties, merits and demerits of the things using the five senses as instruments.

It is very much useful in diagnosing vatha pandu as follows.

Kandal(observation):

By observing the pallor of skin,conjuctiva, nail beds, mucus membrane of lips and tongues.

Karuthal (Inference):

Patients compliants of fatigueness, palpitation, breathlessness on exertion give the clue to the physician in diagnosis of pandu Noi.

Urai(Authority):

In addition to the above manifestation the Naadi shows kabam,kabavatham kaphapitham and vatha kapham which confirms in the diagnosis of vatha pandu. these are quoted in.

“è ‡ ì£«ò£ C«ôŸðù^F™ õ£î ï£®

èô‰F´A™ õJÁ ªð£¼ñ™ èù^î i,è‹

à ‡ ì£«ò£ æƒè£ó... ê^F M,è™

àÁFó†¬ê õ£Œ¾ õLê‰G «î£ì‹

M ‡ ì£«ò£ J¬÷ŠH¼ñ™ «ê£¬ð ð£‡´”

&êîè ï£® Ë™

“î£ùºœ÷ «ê^¶ ñ‰î£Q÷A™ ªõŠ¹

êòl¬÷J¼ñ™ ñ‰î£ó è£ê‹

ßùºÁ...ê‰F Mì«î£ì‹ M,è™

J¼‰ «ó£èƒèóŠð£¡ Móí «î£ì‹

ñ£ù¬ùf˜ Å¬ô Fóœ Mò£F i,è‹

õ¼...ê^F ²õ£ê‹ ªï...ê¬ìŠ¹ É,è‹

ãùºÁƒ è£ñ£¬ô ð£‡´ «ê£¬ð

ã¿²óƒèœ ðô¶,è‹ Mìº‡ì£«ñ”

&êîè ï£®

“Þìñ£ù «ê^¶ñ^F™ H^îï£®

â¿^îµA™ Mìºì«ù i,èº‡ì£‹

Fìñ£ù °O˜ è£„ê™ ñ...êœ «ï£¾^

«îè^F½¬÷„êh¬÷Š H¼ñ™ õ£‰F

Mìñ£ù ªï...ê¬ìŠ¹ ²õ£ê‹ M,è™

ªõ°²óº‹ ï£õø†C ð£‡´ «ó£è‹

Üìñ£ù °õ¬÷ó^î ñFê£ó^î£¡

Üµ A ªõ° ðô «ï£Œ,°^ îìƒè‡ì£«ò”.

&êîè ï£®

“õ£îˆF™ «êˆ¶ñ ñ£A™ õL«ò£´ i,è º ‡ ì£°‹

«ðFˆ¶ˆ î¬ôJ®ˆ¶Š Hí ƒAò ° í ƒèœ

b¶ŸÁ ªñŒªõ Àˆ¶ˆ Fì º ì ù êù... ªê™ô£

«ðFˆ¶ ï£¾ «ð²‹ ªð¼è«õ i,è º ‡ ì£‹”

&Üèˆ Fò˜ ï£®

Enn vagai thervugal:

Enn vagai thervugal namely naadi, sparisam,naa, niram mozhi, vizhi, malam, moothiram are called as the tools in the diagnosis of disease. It is called “Maruthvan Ayutham”.

Therayar quoted this as

“ï£® ðKê‹ ï£ Gø‹ ªñ£N MN

ñô‹ Íˆ F¬ó I¬õ ñ¼ˆ¶õó£»î‹

ªñŒ°P Gø‹ ªî£Q MN ï£ Þ¼‹ðô‹

¬è,°P”.

& «î¬óò˜

Ennvagai thervugal was mentioned in **agathiyar vaidhya vallathi -600 as**

“ªî£,è ÖŸÁ Ü†ìMîŠ ðg†¬ê î¡¬ù

¶ô,è º Á‹ ð‡®î«ó ªîOõî£èŠ

ð°,èKò ï£®¬ò c H®ˆ¶Š ð£¼

ðè˜A¡ø õ£˜ˆ¬î¬òŠ ð£˜ ï£¬õŠð£¼

õ°,èKò «îèªñùˆ ªî£†´Šð£¼

õ÷ñ£ù êgóˆF¡ Gøˆ¬îŠ ð£¼

êA,èKò ñôˆ¬îŠð£˜ êôˆ¬îŠ ð£¼

ê£˜‰î MNî¬ùŠ ð£˜ˆ¶ ªîOõ£Œ, è£«í ".

Agasthiar vaithiya vallathi 600 mentioned the Envagai thervugal as ***attavitha paritchai.***

"ï£®ò£™ º¡«ù£˜ ªê£¡ù ïŸ°P °í ƒè÷£½‹

c®ò MNòù£½‹ G¡ø ï£, °PŠHù£½‹

õ£®ò «ñQò£½‹ ñô«ñ£´ cKù£½‹

Å®ò Mò£F î¡¬ù ²è‹ ªðø õP‰¶ ªê£™«ô".

&Üèˆ Fò˜ ï£®.

Thiruvalluvar says,

"«ï£Œ ï£® «ï£Œ º î™ ï£® Ü¶ î E,°‹

õ£Œ ï£® õ£ŒŠð„ ªêò™"

Naadi:

Naadi is the vitiating element of the body which are vatham,pitham and kapham. Examination of naadi has been recognised as one of the principle means of diagnosis and prognosis of the disease.

Naadi is felt at the lower end of radius, Three fingers that is index, middle and ring finger is placed on the radius which shows vatham, pitham,kapham naadi respectively.Changes in the thiridhosa are felt in naadi.

Edakalai +Abanan -> vatham

Pinkalai + Piranan -> pitham

suzhumunai + samanan -> kapham

the ratio of vatham,pitham, kapham is 1:1/2:1/4 respectively.

In females naadi is felt best on left side and for males on right side.

Naadi is considered as the best tool in the diagnosis of vatha pandu.

In vatha pandu the following types of naadi are seen.

Kapham,kapha vatham, kapha pitham, vatha kapham.

In anaemia naadi is low volume, feeble pulse.

Sparisam (palpitation):

Temperature of the skin, any abnormal growth, hypersensitiveness, dryness of skin, ulcers, oedema are seen in sparisam.

In vatha pandu noi the sparisam is warm.

Naa(Tongue):

The colour changes according to vatham, pitham,kapham, mukkutram are dryness or wet, coated tongue or not, excessive salivation, redness, ulcer, pallor, yellowish discolouration, neoplastic growth, condition of teeth, its colour, condition of the gums, loss of taste, appreciation speech, deviation of the mouth angle movements of the tongue are seen.

In vatha pandu noi tongue was pallor moist and glossy, sense of taste would be altered.

Niram(colour of the skin):

Colour indicating vatham,pitham,kapham and thiridhosa are pallor,yellow discolouration, redness of the skin and bluish discolouration. In vatha pandu the colour of the skin,tongue,nail beds and conjuctiva are pallor.

Mozhi(voice):

Clarity of speech or any disturbances, loud voice, slurring,crying,talk induced by hallucination, breathlessness can be made out.

In vatha pandu the speech is in diminshed voice, breathlessness may occur.

Vizhi(eyes):

Pallor, excessive lacrimation, visual disturbances, suborbital oedema of the eyes are ruled out.

In vatha pandu the conjuctiva is observed which are pallor in this condition.

Malam(faeces):

Quantity, semisolid,colour, odour, froth abnormal consistency , frequency, constipation are observed.

In vatham -ñô , è† ´ ,è¼ˆ î ñô ‹

Hˆ î ‹ & CÁˆ ¶ à † í ˆ ¶ ì ¡ ªê ‹ ¬ ñGø ‹

èð ‹ & Yî ñ£ J¼ , ° ‹

ª î £‰ î ‹ & â‰ î Gø º ‹ Þ¼ , è ô £ ‹

This was quoted as,

è¼ˆ î ñô ð‰ î ñô ƒ è£ô £ ´ ‹ I ˆ î ‹

CÁˆ î º †® í ‹ ªê ‹ ¬ ñ «ê¼ ‹ ª ð£Áˆ ª î £¼ , è£™

Yîñô‰ F™¬ô»ñ£‹ «ê˜‰î ðô «ó£Aò£‹

eîðô‹ â‡ Eøº «ñ.

&Üè^Fò˜ ¬õ^Fò C‰î£ñ E ªõ ‡ ð£400

In vatha pandu noi constipation is usually seen.

Moothiram(urine):

In Agasthiyar vaithiya chinthamani the colour of urine was quoted.

“õ£î«ó£ è‹ ªîO‰î£¡ ñ...êO^î£¡ ñŸ¬øò¶

Yî Â¬ó^ î£˜ðôõ£‹ «ê˜‰î «ï£Œ &«è£îèô£

Í^Fó^F¡ à‡¬ñ ªñ£N‰«î£‹ Þ¬ê ªê£™ô£‹

ï£^Fó^F¡ àœ÷õ¬è ï£‹”.

&Üè^Fò˜ ¬õ^Fò C‰î£ñ E ªõ ‡ ð£ 400

In õ£î‹&c˜ ªîO‰F¼,°‹

H^î‹&ñ...êœ Gø‹

èð‹&Yî‹ èô‰¶ è£íŠð´‹

ªî£‰î‹&Þ¬õ èô‰F¼,°‹

Quantity, colour,odour, frothy,frequency,deposits,presence of abnormal constituents such as sugar, albumin etc... are seen.

It contains two phase in diagnosing they are

- Neerkuri (colour of the urine)
- Neikuri (oil examination)

Collection of urine:

“Ü¼‰¶ ñ£Póî º ‹ ÜM«ó£îñî£Œ

Üçè™ Üô˜î™ Üè£ô× ¡ îM˜‰îöŸ

°ŸøùõF¼‰ àøƒA ¬õè¬ø

Ý®,èôêˆ î£M«ò è£¶ªðŒ

ªî£¼º Ã˜ˆî, è¬ô,°† ð´ cK¡

Gø,°P ªïŒ,°P G¼IˆîèÌ«ù.”

The patient must take well cooked food, propotionate to the degree of his appetite at apporopriate time on the previous day.After having sleep at night the urine is collected on the early morning of the next day in a glass container and to be examined within hours.

Neerkuri:

“õ‰î c˜,èK â¬ì ñ í ‹ ¸¬ó â...êªô¡

¬ø‰Fò½÷õ¬õ ò¬ø°¶ º¬ø«ò”

In neerkuri colour, odour, specific gravity, forthy, quantity, deposits were observed.

Colour:

Yellow,red,green, black, crystal, smoky, copper.

Manam(Smell):

Pleasant, foul smelling, honey, fruit and flesh smell.

Nurai(Forthy):

Forth with or without colouration.

Enjal(Quantity):

Amount of urine expelled and any deposits in urine are observed.

Frequency:

Increased or decreased frequency, dribbling, incontinence of urine.

And the specfic gravity of urine are observed.

Neikuri:

“Gø , ° P , ° ¬ó ˆ î G¼ñ£ù cKŸ

Cø , è ª õ ‡ ª í «ò£˜ CÁ ¶ O ï ´ M ´ ˆ

ª î ¡ Áø‰ Fø‰ª î £L «òè£è ¬ ñ ˆ î F

Q ¡ øF õ ¬ ô «ð£‹ ª ï P õ N òP¾‹

ª ê ¡ ø ¶ ¹ è½… ª êŒF ¬ ò » í «ó”.

The speciman is kept open in a glass bowl being exposed well in sunlight. Not distrubed by wind then add one drop of gingelly oil by a glass rod observe about the spreading of the oil drop on the surface.

vatham - Üóª õ ù c ‡ ì ¶

pitham - Ý N«ð£™ðóM ò ¶

kapham & º ˆ ª î £ˆ ¶ G ¡ ø ¶

In vatha pandu noi snake like spreading was observed.

Beside enn vagai thervugal disease can also be diagnosed by other methods namely thinaigal, paruvakalangal,uyir thathukal,udal thathukal. Hence a through knowledge about the disease can be attained systematically and properly in siddha system of medicine. A combination of all these diagnostic creteria is very helpful to

attain a proper diagnosis with a full complete entity based on basic principles of siddha science.

Thinaigal:

Thinai (or) Nilam is classified into five types depending on the surrounding, vegetation,landscape and ecological state . Study of thinai is very much necessary.

Kurinji(Mountain and its surrounding):

“ ° P...C õ¼ GôˆFŸ ªè£Ÿø º ‡ ® óˆî ‹

àP...C õ¼²óº º ‡ ì£‹ & ÜPë¼¬ó,

¬èò«ñ îƒ° îóˆ î£¬ñõ™¬ô »ƒèF,°‹

äò«ñ îƒ° ñP”.

Liver disease, Fluorosis ,Haematological disorder are common in this land.

Mullai(Forest and its surrounding):

“ º ™¬ô GôˆîÒ¬ñ ºKG¬ó «ñMÂñš

ªõ™¬ô G¬ôˆî HˆîÒ ªñƒ°Áƒè£¡ & Ü™ôªòQ™

õ£îªñ£N ò£îî µ ‡ ñ¡Âñ¬õ õN«ï£ŒŠ

«ðîªñ£N ò£î¬øòŠ H¡¹”.

Vatha and pitha disease are common in this land.

Marutham(Field and its surrounding):

“ñ¼îGô ï¡m˜ õ÷ªñ£¡¬ø, ªè£‡«ì

ªð£¼î Gô ñ£Fò «ï£Œ «ð£,°ƒ & è¼îGôˆ

î£Póî... Åö õ¼ˆ¶õ ªó¡ø£Ÿ H E ªò™

ãPóî... Å›M,° I ™.”

It is a suitable land for the mankind for healthy living.

Neithal(Sea, seashore and its surrounding):

“ª ïŒî Q ô «ñ½õ˜Š¬ð cƒè£ ¶P Â ñ¶

ªõŒî Q ô «ñ î ƒ ° i ì£ ° ‹ & ª ï£Œb ¡

ñ¼ƒ ° ì ¬ ô º ,è£ ,A õ™½ÁŠ¬ð i , ° ƒ

è¼ƒ ° ì ¬ ô , WNøƒ ° ƒ è£ ‡ .”

& ðî£˜ˆ î ° í C‰î£ñ E .

Increased kapham causes oedema, elephantiasis, liver disease are common.

Palai(Dessert and its surrounding):

“ð£¬ ô Gô ‹ «ð£Ÿ ðì¬óŠ HøŠH ,è

«ñ¬ ô Gô eò£¶ MKˆ îŸ° & «õ¬ ôGô

ºŠH E , ° I ™ô£‹ º ¬ø«òòõŸø£ô£‹

âŠH E , ° I ™ô£ ñçª î ¡.”

& ðî£˜ˆ î ° í C‰î£ñ E .

Vatha, pitha, kapham and its related disease’s are common in this land.

In all five thinai vatha pitham is probable of occurance.

Paruvakalangal(seasonal variation):

Earth take 12 month’s to rotate sun according to this year is divided into six season they are

Kalam	**Tamil months**	**English months**
Kaarkalam	Avani,Purattasi	Monsoon
Koothir kalam	Ipasi, karthigai	Autumn

Munpani kalam	Margazhi, thai	Winter
Pinpani kalam	Maasi, panguni	Prevernal
Ilavenil kalam	Chithirai, vaigasi	Spring
Muthuvenil kalam	Aani, aadi	Summer

Each season consists of 2 months each.

In all six seasons vatha pandu probaly occurs.

Udal vanmai(Immunity):

Udal vanmai is classified into three types they are

- Iyarkai vanmai
- Seyarkai vanmai
- kaala vanmai

Iyarkai vanmai:

Natural immunity of the body caused by mukkutram right from birth onwards.

Seyarkai vanmai:

Improving the health by intake of nutritious food, physical activites and by medicine (or) Immunization.

Kaala vanmai:

Development of immunity according to age and environment.The alteration in udal vanmai creates more possibilities to vatha pandu noi.

Uyir thathukal (mukkutram):

Uyir thathukal are

- vatham
- pitham
- kapham

Which acts as an vital elements and it is the functional units of the body.

Vatham:

This is a kinetic energy,which enfluence all motions. It is located in abanan, edakalai,motion,spermatic cord,pelvic bone,nerves, joints and muscles.Vatham represents vayu,mind,dryness, pain, flatulence,sensitiveness,lightness and air.

They are classified into ten types,they are

1.Piranan(uyirkaal)

This is called as heart center. Piranan means the forward or primary air forces it controls breathing knowledge,mind and five sensory organs.

2. Abanan(Keelnokunkaal)

This corresponds to the pelvic plexus and is the seat of kundalini or material energy situated in the naval region and expels faecal matter,urine , menstrual flow,sperm and ovum constricts and spincter carries ingested food extracts to the respective places.

3. Viyanan(paravukaal)

This corresponds to the nasociliary plexus viyanan means the diffusive air it is responsible for the nutrition and movement of all movable and immovable parts of the body causes the feeling of sensation.

4. Uthanan(mel noku kaal)

Corresponds to pharyngeal plexus present in throat region and controls

breathings and speech uthanan means moves upward air force. It is responsible for nausea vomiting and erucation.

5. Samanan (nadukaal):

Corresponds to solar plexus. Samanan means equalizing air it regulates all other four main vital air forces and it aids proper digestion.

6. Naagan :

Responsible for higher intellectual function, causes opening and closing of eye lids.

7. Koorman:

Responsible for vision and yawning.

8. Kirukaran:

Lies in the tongue, salivary and nasalsecretion causes hunger, concentration of the mind on the particular thing sneezing and responsible for taste sensation.

9. Devathathan:

Responsible for lazziness,sleeping and anger.

10.Dhananjayan:

Produce bloating of the body and escape on third day after death. In case of vatha pandu

S.No	Names	Features
1.	Abanan	Expells stools
2.	Viyanan	It carries nutrients all over the body
3.	Udhanan	Vomiting
4.	Samanan	Due to other vayus are affected

5.	Kiruharan	Appetite affected
6.	Thevathathan	Tiredness and sleep is affected

These vayus are affected in vatha pandu.

Pitham:

Pitham represents gastic juice,bile,energy, heat, anger and irritation. Located in urinary bladder,heart,head, pingalai umbilicus, abdomen,piranan ,blood,sweat,skin and eyes.

They are classified into five types

Name	**Features**
Anal pitham (Gastric juice)	Give appetite and helps in digestion.
Pirasagam(Bile)	Gives complexion to the skin
Ranjaga Pitham(Haemoglobin)	Which colours the blood
Aalosagam(Aqueous humour)	Brightens eyes and helps in vision
Saathaga pitham (Life energy)	Controls whole body

In case of vatha pandu noi

Anal pitham,Ranjagam, pirasagam and sathaga pitham is affected.

Kapham:

Kapham represents feeling of cold, heaviness, running nose, passing of muscoid discharge and also saliva.

Located in samanan, seminal fluid ,head,tongue,fat, bone marrow, blood, nose, chest, nerves, bones, large intestine, stomach and pancreas.

classified into 5 types they are

Name	Features
Avalambagam	In helps the heart in pumping and controls other four kapham.
Kilethagam (saliva)	Present in mucus secretion of mouth and in mouth, makes food wet and helps in mastication.
Pothagam(Lymph)	Located in tongue and saliva and gives taste sensation.
Tharpagam (cerebrospinal fluid)	This is located in head controls cerebrospinal fluid keeps head and eyes cool.
Santhigam(synovial fluid)	Located in joints which lubricates and aid free movements of the joints.

In vatha pandu noi

Kilethagam, pothagam, Avalabagam are commonly affected.

UDAL THATHUKAL:

Udal thathukal are seven in number which constitutes the entire body.

1. Saaram: Contains nutrients from digested food and nourishes all tissue, organs and system.

2. Senneer: Oxygenates all tissues and vital organs and maintains life.

3. Oon: Gives structure and shape to the body responsible for the movements of the body.

4. Kozhuppu: Lubricates the joints and facilitates their functions.

5. Enbu: Protects all the internal organs and gives structure to the body.

6. Moolai: Located in between the core of the bones and gives strength to bones.

7. Sukkilam / Suronitham: Meant for reproduction incase of vatha pandu Saaram, senneer, oon,kozhuppu are affected mainly.

Noi nithanam:

Progosis:

According to yugimuni in yugi vaithia chinthamani vatha pandu noi is curable.

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In piramegam, vathasoolai, neerizhivu ,gunmam, shayam, sanni, kamalai, pandu,sobai, kapha rogangal,manjal noi,paithiya rogam if diarrhoea occurs it becomes worsen.

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The above verse was quoted in sathaga naadi this denotes in paandu noi and in other certain diseases if the combination of emaciation,dyspnoea and hiccough occurs it causes poor progonosis of the disease.

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The above quote denotes that scanty micturation in pandu noi causes death of the patient.

In the above condition the prognosis of vatha pandu noi may be poor and it becomes incurable causes death.

Maruthuvam:

Our siddha treatment is not only for complete healing but also for the prevention and rejuvenation. This is said as

Kappu(Prevention)

Neekam(Treatment)

Niraivu(Restoration)

Siddha system has uneqivocally stated that even during the time of conception, some defects creep into the fertilised embryo. The defects from the basis for the manifestation of certain constitutional disease later on during the existence of the individual.

Diseases are produced by the unequilibrium of three thathus which may be due to various causes like diet, lifestyle, mental and physical activities .It is essential to know the cause for the disease the nature of the patient and the severity of illness and the seasons and the time of the occurance of the disease.

Line of treatment

The aim is to normalise the vitiated thathus and affected raktha thathu. Effective medicinal preparations have to be administered in the beginning itself to raise the raktha thathu to attain its normal functions.

Diet

Siddha system lays a great importance on the observation of rules regarding diet in everyday life because the siddha system has rightly realised that the basic factor of the body is food, that is Annamaya kosam is the first among the five kosams constituting our physical and mental existence to prevent the occurrence of the disease. elaborate inference regarding food item is our daily diet is given in the text book of siddha.

Thirumoolar says

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Herbs, Metals and Minerals commonly used for pandu noi

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Diet Regimen for pandu noi

Consuming unbalance and incompatible diet is considered to be the prime causative factor for upsetting the threedosha balance leading to manifestations of various ailments regarding diet regimen in vatha pandu the following food items are recommended.

Keerai vagaikal (greens)

Greens like Karisalai, Ponnanganni, Arukkeerai, Sirukeerai, Murungai Keerai and manithakkali keerai having haematinic property may be given daily.

Kaikarikal (vegetables)

Kathiri pinju, Avarai pinju, Murungai pinju, Vazhai kachal may be given along with other food stuffs.

Pazhavakaigal (fruits)

Pereechai, Orange, Kodimunthiri, Naral, Apple, Atthi are also rich sources for iron.

Easily digestable foods like kanji, Mutton soups, bone soups must be given in acute stages of vatha pandu noi. The goat liver soup is given with orange (or) apple (or) grape juice to these patients.

After the normal appetile is restored properly prepared flesh of kadai, kowthari, and udumbu can also be given. They tone up the body in the debilitaling condition and improve “Raktham” formation.

If oedema is present barley Kanjee prepared with vellarivithu. Poosani vithu can be given. This will act as diuretic and reduce oedema.

When the symptoms were improved the patients were advised to practice Yoga and pranayama according to their physical and mental conditions.

MODERN ASPECT

INTRODUCTION

Nutritional anemia is a global problem of immense public health significance, affecting persons of all ages and economic group, it is more common among preschool children's, who are taking poor diet and pregnant women's. It is ranked as the commonest chronic woe of mankind. Around 30% of the total world population is anaemic and half of these, some 600 million people have iron deficiency the highest prevalence is in pre school children's 47.4% and the lowest prevalence is in men 12.7% about 55% of pregnant women's are affected by this. Even affluent section suffers from anemia due to ignorance of food. Actually anemia is the condition in which the haemoglobin level in the blood is below the normal range and this is discussed in various views.

The Blood and its components

Blood is the connective tissue in the fluid form, it is considered as the fluid of life because it carries oxygen from lungs to all parts of the body and carbon dioxide from all parts to the lungs. The blood is the red fluid of alkaline reaction and is salty in taste. The whole blood volume of infants has been reported as 90ml/kg.

Blood consists of blood cells which are called as formed elements and the liquid portion known as plasma. The cell make up 40-45% of the total amount of

the blood and plasma makes about 55-60% the cells consists of 90% of haemoglobin and 10% of stroma.

Blood cells:

Three types of cells are present in blood they are

1. Red blood cells or erythrocytes

2. White blood cells or leucocytes

3. Platelets or thrombocytes

Erythocytes or Red Blood Cells:

Red blood cells are the non-nucleated formed elements in the blood. The red colour of these cells is due to the presence of the colouring matter haemoglobin in these cells.

The red blood cell count ranges between 4 to 5.5millions/cu mm of blood.

In adult males it is 5 millions/cu mm of blood.

In adult females it is 4.5 millions/cu mm of blood.

Shape and Size of Red Blood Cells:

Normal red blood cells, are biconcave discs having a mean diameter of about 7.8micrometer and a thickness at the thickest point of 2.5 micrometer and

in the center of 1micrometer or less. The average volume of the red blood cell is 90 to 95 cubic micrometers.

The shapes of red blood cells can change remarkably as the cells pass through capillaries. Actually, the red blood cell is a “bag” that can be deformed into almost any shape. The normal cell has a great excess of cell membrane for quantity of material inside, deformation does not stretch the membrane greatly and consequently, does not rupture the cells, as would be the case with many other cells.

Functions of the blood:

* Transport of oxygen from lungs to the tissues to utilize metabolic process
* Carry CO_2 from tissues to lungs
* Transports nutrients, other metabolites, hormones from the source to the site of usage, action or exertion.
* Responsible for maintain the thermoregulatory mechanism in the body
* Carries materials that clot blood preventing its loss from a ruptured blood vessel
* Blood communicates between the cells of different parts of the body

Erythropoiesis:

Is the process which involves the origin, development and maturation of erythrocytes.

Site of Erythropoiesis:

Areas of the body that produce red blood cells

* In the early few weeks of embryonic life – yolk sac
* During the middle trimester of gestation – liver, spleen and lymph nodes
* Later part of gestation and after birth – red bone marrow
* Up to the age of five – red bone marrow of all bones
* From the age of six to twenty – red bone marrow of proximal end of long bones and all the membranous (flat) bones.
* After the age of twenty – from membranous bones like vertebra, sternum, ribs, scapula, iliac bones and skull bones and from the end of long bones.
* During bone disorders the RBC are produced in spleen

Genesis of Red Blood Corpuscles:

In the bone marrow there are cells called pleuripotent hemopoietic stem cells (PHSC) from which all the cells in the circulating blood are derived. The larger of reproduced stem cells differentiate to form the other cells, even though they have

already become committed to a particular line of cells and are called committed stem cells.

The different committed stem cells will produce colonies of specific type of blood cells. Therefore committed stem cells that produces colony forming unit blast (CFU – B) and then erythrocytes are produced from this are called colony – forming unit – erythrocytes (CFU – E).

Growth and reproduction of the different stem cells are controlled by multiple proteins called growth inducers the another set of proteins are called differentiation inducers whose function is differentiation of the cells.

Stages of Erythropoiesis:

Pluripotent hemopoietic stem cells

Colony forming blastocytes

Colony forming unit erythrocytes (CFU-E)

Proerythrocyte
(first cell that belonging the RBC series unipotential)

Early normoblast or basophil erythroblast
(begins synthesis of heamoglobin)

Intermediate normoblast

Late normoblast
(with small nucleus and more haemoylobin orthochromic erythroblast)

Reticulocyte
(small amount of basophilic reticulum is present)

Erythrocytes
(matured red blood corpuscles)

Regulation of Erythropoiesis:

Tissue oxygenation is the basic regulator of red blood cells production. Any condition that causes the quantity of oxygen transported to the tissues to decrease (hypoxia) ordinarily increases the rate of red blood cells production. Main conditions being very high altitudes, anemia, prolonged cardiac failure and lung diseases.

Erythropoietin is the circulating hormone which is the principle factor that stimulates red blood cells production. It is a glycoprotein with a molecular weight of about 34,000. Hypoxia causes marked increase in erythropoietic production.

Kidney produce about 90% of all erythropoietin in a normal person, the remaining is formed from the liver. Epinephrine, norepinephrine and several of the prostaglandins stimulates erythropoietic production.

Substances Necessary for the Formation of Red Blood Cells

1. Protein

Hemoglobin synthesis depends on adequate supply of the proper amino acids for the synthesis of globin and clinical, experimental evidence show that anemia can be caused are aggregated by protein deficiency.

2. Iron

About 15mg of iron is needed daily for growing children and women. This is essential for the proper hemoglobination of red blood cells in their later stages of maturation i.e. red corpuscles. A deficiency of iron causes hypochromic microcytic anemia.

3. Copper

Traces of copper are essential for the utilization of iron in the synthesis of hemoglobin.

4. Manganese

Probably act as a catalyst.

5. Vitamins

Vitamin B_{12} and folic acid are the bone marrow stimulants. Vitamin B_{12} along with the intrinsic factor present in the stomach act as proerythroblastic stage. So

that if it is absent the maturation is defected to the megalocytic series of cell instead of forming normoblast.

Vitamin A. vitamin C is also a bone marrow stimulant acting at any particular stage of maturation. Its deficiency causes normocytic type of anemia.

6. Internal secretions

Thyroxine is a general metabolic stimulant which increases the tissues activity and oxygen demand, to this demand the bone marrow response adenocorticotrophic hormone and cortisones appear to have some relationship with the red cell formation.

7. Bile

Bile appears to help the absorption of iron and utilization of iron by the tissues.

HEMOGLOBIN

Hemoglobin is the colouring matter of RBC. It is a chromoprotein forming 95% of dry weight of RBC and 30 to 34% of wet weight. The function of hemoglobin is to carry the respiratory gases. It also acts as a buffer.

Structure of Hemoglobin:

Hemoglobin is a conjugated protein. It consists of a protein combined with an iron containing pigment. The protein part is globin and iron containing pigment is heme. Heme also forms a part of structures of myoglobin (oxygen binding pigment in muscles) and neuroglobin (oxygen binding pigments in brain). Iron is normally present in ferrous form. The pigment part is called porphyrin. It is formed by four pyrole rings. The rings are attached to one another by methane bridges. Globin consists of four polypeptide chains. Two are alpha and two are beta. The molecular weight of hemoglobin is 68,000.

Synthesis of Hemoglobin:

Synthesis of hemoglobin actually starts in proerythroblastic stage. However hemoglobin appears in the intermediate normoblastic stage only. The production of the hemoglobin is continued until the stage of reticulocyte. The Heme portion of hemoglobin is synthesized in mitochondria from acetic acid and the glycine.

The sequence of events in synthesis of hemoglobin:

* During Krebs cycle, the acetic acid is converted into succinyl CoA.

* Two molecules of succinyl CoA combine with two molecules of glycine to form pyrole compound.

* Four pyrole compounds combine to form protoporohyrin

* Protoporphyrin is of many types. Only protophyrin IX is involved in the formation of heme molecule by combining with iron.

* Each heme molecule combines with one globin molecule to form hemoglobin.

Combination of Hemoglobin with Oxygen:

The primary function of hemoglobin is to combine with oxygen in the lungs and then to release this oxygen readily in the tissue capillaries where the gaseous tension of oxygen is much lower than in lungs. One gram of hemoglobin combines with about 1.39 ml of oxygen.

Destruction of Hemoglobin:

After the lifespan of 120 days, the RBC is destroyed in the reticuloendothelial system particularly in spleen and the hemoglobin is released into plasma. Soon, the hemoglobin is degraded in the reticuloendothelial cells and split into globin, iron and porphyrin.

Globin is utilized for the resynthesis of hemoglobin. Iron is stored in the body as ferritin and hemosiderin, which are reutilize for systhesis of new hemoglobin. Porphyrin is convered into a green pigment called biliverdin.in human being, most of the biliverdin is converted into a yellow pigment called biriubin. Bilirubin and biliverdin are together called the bile pigment.

Normal Values:

Average hemoglobin content in blood is 14 to 16 gms%. However the value varies depending upon the age and sex of the individual.

Age

At birth – 25 gms%

After third month – 20 gms%

After one year – 17 gms%

From puberty onwards – 14 to 16 gms%

Sex

Adult males – 15 gms%

Adult females – 14.5 gms%

Packed cell volume (haematocrit value)

Men – 40 to 50 %

Women – 35 to 47 %

Infants – 44 to 62 %

Children (one year) – 36 to 49 %

Children (10 to 12 year) – 37 to 44 %

Mean Corpuscular Volume (MCV)

Adult – 76 to 96 Cubic microns

Infants (3 month) – 83 to 110 Cubic microns

Children (1 year) – 77 to 101 Cubic microns

Children (10 to 12 years) – 77 to 95 Cubic microns

Mean corpuscular diameter

Adult -6.6 to 7.7 microns

Mean Corpuscular Hemoglobin (MCH)

$$MCH = \frac{Hb\ Concentration\,(grams\ per 100ml)}{RBC\ Count\,(Million\ \ per\ \ ml)}$$

Adults – 27 to 32 pico grams

Mean Corpuscular Hemoglobin Concentration (MCHC)

$$MCHC = \frac{Hb\ in\ \ grams\ per 100ml}{Volume\ of\ Packed\ \ Cell\ per\ 100\,ml}$$

The average value is 35 ± 3 percent.

IRON METABOLISM

Iron is important for the formation of hemoglobin, myoglobin and other substance like cytochrome ,cytochrome oxidase, peroxidase and catalase.

Normal Value and Distribution of Iron in the Body:

The total quantity of iron in the body is about 4 grams. The approximate distribution of iron in the body is as follows:

In the hemoglobin : 65 to 68 %

In the muscle as myoglobin : 4 %

As intracellular oxidative heme compound : 1%

In the plasma as transferring : 0.1%

Stored in the reticuloendothelial system : 25 to 30 %

Dietary Iron:

Dietary iron is available in two forms called heme and nonheme. Heme iron is present in fish, meat and chicken. Non heme iron is available in vegetables, grains, and cereals.

Rich : Liver, egg yolk, oyster, dry beans, dry fruits, yeast.

Medium : Meat, chicken, fish, spinach, banana, apple,

Poor : Milk and its products, root vegetables.

Daily Requirement of Iron:

Adult male	:	0.5-1mg
Adult female	:	1-2mg
Infants	:	60µg/kg
Children	:	25µg/kg
Pregnancy women	:	3 – 5mg

Iron requirement (mg) = 4.4× body weight× Hb deficit

Absorption of Iron:

Iron is mainly absorbed from the small intestine. It is absorbed through cells by pinocytosis and transports into the plasma. Bile is essential for the absorption of iron.

Transport and Storage of Iron:

Immediately, after absorption into the blood, iron combines with a â globin called apotransferrin to form transferrin and is transported in this form in the plasma. Iron combines loosely with the globin and can be released easily at any region of the body.

Iron is stored in large quantities in the reticuloendothelial cells and liver hepatocytes. In other cells also, it is stored in small quantities. In the cytoplasm of the cell, iron is stored as ferritin in the large amount. Small quantity of iron is also stored as hemosiderin.

Daily Loss of Iron:

In males, about 1 mg of iron is excreted every day through feces. In females, the amount of iron loss is very much high. This is because of the menstruation.

One gm of hemoglobin contains 3.34 mg of iron. Normally, 100 ml of blood contains 15 gm of hemoglobin and about 50 mg of iron (3.34 × 15). So, if 100 ml of blood is lost from the body, there is loss of about 50 mg of iron. In females, during every menstrual cycle, about 50 ml of blood is lost by which 25 mg of iron is lost. That is why the iron content is always less in females than in males.

Iron is lost during hemorrhage and blood donation also. If 450 ml of blood is donated, about 225 mg of iron is lost.

Regulation of Total Blood Iron:

Absorption and excretion of iron are maintained almost equally under normal physiological condition. When the iron storage is saturated in the body, it automatically reduced the further absorption of iron from the gastrointestinal tract by feedback mechanism.

The factors which reduce the absorption of iron are:

1. Stoppage of apotransferrin formation in the liver, so that, the iron could not be absorbed from the intestine.

2. Reduction in the release of iron from the transferring so that, transferring is completely saturated with iron and further absorption is prevented.

ANEMIA

Anemia refers to a state in which the level of haemoglobin in the blood is below the reference range appropriate for age and sex. It is one of the common blood disorders it is refers to reduction in Red blood cell count, Hemoglobin content, packed cell volume.

Generally anemia occurs because of:

1. Decreased production of RBC
2. Increased destruction of RBC
3. Excess loss of blood from the body

All these incidents are caused either by inherited disorders or environmental influences such as nutritional problem, infection and exposure to drugs or toxins. In these conditions, the change is noticed not only in hemoglobin content but also in the morphologic features of the RBC.

Aetiology:

1. Blood loss
2. Impaired red cell production
3. Inadequate supply of nutrients essential for erythropoiesis such as,

a. Iron deficiency

b. Vitamin B_{12} deficiency

c .Folic acid deficiency

d. Protein-caloric malnutrition

e. Other less common deficiency

4. Depression of erythropoietic activity

5. Anemia associated with chronic disorders such as

a. Infection

b. Connective tissue disorder

c . Inflammatory disorders

d. Disseminated malignancy

6. Anemia associated with renal failure

7. Anemia due to replacement of normal bone marrow by

a. Leukemia

b. Lymphoma

c . Myeloproliferative disorders

d. Myeloma

Classification of Anemia

Anemia is classified by two methods

A. Morphological classification

B. Etiological classification

Morphological Classification:

Morphological classification depends upon the size and colour of RBC. Colour is determined by quantity of hemoglobin, according to this anemia is classified into four types.

1. Normocytic Normochromic Anemia

The size and heamoglobin content of RBC are normal but the number of RBC is less.

2. Macrocytic Normochromic Anemia

The RBC is larger in size with normal hemoglobin content, the RBC count decreases.

3. Macrocytic Hypochromic Anemia

The RBCs are larger in size. The hemoglobin content in the cell (MCH) is less so the cells are pale in colour.

4. Microcytic Hypochromic Anemia

The RBCs are smaller in size and the hemoglobin content (MCH) is less.

Etiological Classification

On the basis of etiology anemia is classified into five types:

1. Hemorrhagic anemia.
2. Hemolytic anemia.
3. Nutritional deficiency anemia.
4. Aplastic anemia.
5. Anemia of chronic disease.

1. Hemorrhagic anemia:

Anemia due to hemorrhage is known as hemorrhagic anemia. It occurs both in acute and chronic hemorrhagic condition. Hemorrhage occurs in conditions like accident, ulcer, excessive uterine bleeding, purpura and hemophilia.

Acute Hemorrhage – it refers to sudden loss of large quantity of blood as in the case of accident. Within about 24 hours of hemorrhage, the plasma portion of blood is replaced. However the replacement of RBC does not occur quickly and it takes at least 4-6 weeks. So with less number of RBCs hemodilution occurs. However morphologically the RBCs are normocytic normochromic. Decreased RBC count causes hypoxia which stimulats the bone marrow to produce more number of RBC. So, this condition is corrected within 4 to 6 weeks.

Chronic Hemorrhage – It refers to loos of blood by internal or external bleeding over a long period of time. It occurs in conditions like peptic ulcer, purpura, hemophilia and Menorrhagia.

Due to continuous loss of blood, lot of iron is lost from the body causing iron deficiency. This affects the synthesis of hemoglobin resulting in less hemoglobin content in the cell. The cell also becomes small. Hence the RBCs are microcytic and hypochomic.

2. Hemolytic Anemia

Hemolysis means destruction of RBCs. Hemolytic anemia occurs because of excess destruction of RBCs.

Causes of excess hemolysis are:

1. Liver failure
2. Renal disorder
3. Hyperspleenism
4. Burns
5. Infection like malaria and septicemia
6. Poisoning chemical substances like lead, coal and tar
7. Presence of isoagglutinins like anti Rh
8. Congenital or acquired default in the shape of RBCs.

When the shape is abnormal, RBCs become more fragile and hemolysis occur easily. It occurs in two inherited conditions called sickle cell anemia and thalassemia.

Sickle cell anemia

It is a congenital anomaly and found mostly in blacks. It is due to the abnormal hemoglobin called hemoglobin S (normal adult hemoglobin is hemoglobin A). In this, alfa chains are normal and beta chains are abnormal. The molecules of hemoglobin S polymerize into long chains and precipitate inside the cell. Because of this, RBCs attain sickle (crescent) shape and become more fragile leading to hemolysis.

In children, hemolyzed sickle cells aggregate and block the blood vessels leading to infraction (stoppage of blood supply). The infraction is common in small bones. The infracted small bones in hand and feet results in varying length in the digits. This condition is known as hand and feet syndrome. Jaundice also occurs in these children.

Thalassemia

It is also known as Cooley's anemia or Mediterranean anemia. It is more common in Thailand and to some extent in Mediterranean countries. This type of anemia is due to the inherited anomalies of hemoglobin.

Thalasemia is of two types:

1. α **thalassemia**

2. β **thalassemia**

The β **thalassemia is very common among these two:**

In normal hemoglobin, the number of α and β poly peptide chains is equal. In thalassemia the production of these chains become imbalanced because of defective synthesis of globin genes. This causes the precipitation of in poly peptide chains in the immature RBCs leading to distributions in process of erythropoiesis. The precipitation also occurs in mature red cells resulting in hemolysis.

The α thalassemia occurs in fetal life or infancy in this α chains are less, absent or abnormal with excess of γ chains. This leads to defective erythropoiesis and hemolysis. The infants may be stillborn or may die immediately after birth.

In â thalassemia â chain are less in number, absent or abnormal and there is on excess of á chains. á chains precipitate causing defective erythropoiesis and hemolysis.

3. Nutrition Deficiency Anemia

Nutritive substances such as iron, proteins and vitamins like C,B_{12} and folic acid are necessary for erythropoiesis. The deficiency of these substances leads to nutrition (nutritional) deficiency anemia.

Iron Deficiency Anemia:

Iron deficiency anemia is most common type of anemia. It develops due to inadequate availability of iron for hemoglobin synthesis. The RBCs are microcytic and hypochromic.

Causes of Iron Deficiency Anemia are

1. Loss of blood

2. Decreased intake of iron

3. Poor absorption of iron from intestine

4. Increased demand for iron in condition like growth and pregnancy

The feature of iron deficiency anemia are brittle nails, spoon shaped nails (koilonychias), brittle hair, atrophy of papilla in tongue and dysphagia.

Protein Deficiency Anemia:

Due to deficiency of protein the synthesis of hemoglobin is reduced. The RBCs are macrocytic and hypochromic.

Pernicious Anemia or Addisons Anemia:

It is due to atrophy of the gastric mucosa because of autoimmune destruction of parietal cells, the gastric atrophy results in decreased production of intrinsic factor and poor absorption vitamin B_{12} which is the maturation factor of RBC. The RBCs are larger and immature with almost normal hemoglobin content. So, cells are macrocytic and normochromic.

Before knowing the cause of this anemia, it was very difficult to treat the patients and the disease was considered to be fatal. So, it was called pernicious anemia. The synthesis of hemoglobin is almost normal in this type of anemia.

Pernicious anemia is common in old age and it is more common in females than in males. It is associated with other autoimmune diseases like disorders of thyroid gland, Addison's diseases, etc. The characteristic feature if this type of anemia are lemon yellow colour of skin (due to anemic paleness and mild jaundice) and red sore tongue. Neurologically disorders such as paresthesia (abnormal sensations like numbness, tingling, burning etc) progressive weakness and ataxia (muscular incoordination) are also observed in extreme conditions.

Megaloblastic anemia

Megaloblastic anemia is due to the deficiency of another maturation factor called folic acid. Here the RBCs are not matured. The DNA synthesis is also defective so the nucleus remain immature. The RBCs are megaloblastic and hypochromic.

The feature of pernicious anemia appear in megaloblastic anemia also. However,neurologically discords may not develop.

4. Aplastic Anemia

Aplastic Anemia is due to the disorder of red bone marrow. The red bone marrow is reduced and replaced by fatty tissues. It occurs in conditions like repeated exposure

to X-ray or gamma ray radiation and by the presence of bacterial toxins, quinine, gold salt, benzene, radium etc., it is common in tuberculosis and viral infections like hepatitis and HIV infection. The cells are normocytic and normochromic.

5. Anemia of Chronic Diseases

Anemic of chronic diseases is the second common type of anemia (next to iron deficiency anemia). It is characterized by short life span of red cell caused by disturbance in iron metabolism or resistance to erythropoietin action. Anemia develops after few months of sustained diseases.

Common cause of this type of anemia is:

i. Non-infectious inflammatory diseases such as rheumatoid arthritis (chronic inflammatory auto immune disorder affecting joints)

ii. Chronic infection like tuberculosis (infection caused by mycobacterium tuberculosis) and abscess (collection of pus in the infected tissue in lungs)

iii. Neoplastic disorders (abnormal and disorganized growth in tissue or organ) such as Hodgkin's diseases (type of malignancy involving lymphocytes) and cancer of lung and breast.

The red cells are generally normocytic and normochromic in this type of anemia. However, in progressive disease associated with iron deficiency the cell become microcytic and hypochromic.

Types of anemia	**Causes**	**Morphology of RBC**
Hemorrhagic anemia	Acute loss of blood Chronic loss of blood	Normocytic, Normochromic Michrocytic, Hypochromic
Hemolytic anemia	1.Liver failure 2. Renal disorder 3. Hypersplenism 4. Burns 5. Infections- malaria and septicemia 6. Poisoning by chemicals like lead, coal and tar 7. Isoagglutinins- anti Rh 8. Congenital or acquired default in the shape of RBCs	Normocytic, Normochromic Sickle cell anemia-sickle shape Thalassemia: small and irregular
Nutritional deficiency anemia	Iron deficiency Protein deficiency Vitamin B_{12} deficiency Folic acid deficiency	Microcytic Hypochromic Macrocytic, Hypochromic Macrocytic, Hypochromic/ Normochromic Megaloblastic, Hypochromic
Aplastic anemia	Bone marrow disorders	Normocytic, Normochromic
Anemia of chronic diseases	1.Non-infectious inflammatory diseases 2. Chronic infection 3. Neoplastic disorder	Normocytic, Normochromic

Iron Deficiency Anemia

Iron deficiency is the most common nutritional deficiency in the world and the most common cause for anemia world wide the factors which influence the prevalence of anemia include socio-economic status, dietary patterns, the degree of urbanization, educational background and accessibility to health care facilities prophylaxis programmes and the prevalence of worm infestations in the population.

Etiology:

The etiology varies with the age, sex and country of residence of the patient

I. Increased Blood Loss:

1. Uterine

Eg: a) Excessive menstruation in reproductive years

b) Repeated miscarriages

c) At onset of menarche

d) Post menopausal uterine bleeding

e) Uterine cancer

2. Gastrointestinal

a) pepticulcer

b) Hemorrhagic gastritis

c) Gastric carcinoma

d) Colonic carcinoma, Haemorrhoids

e) Hook worm or pin worm diseases

f) Oesophagal varices

g) Hiatus hernia

h) Chronic aspirin ingestion

i) Ulcerative colitis and crohn diseases

ii) Diverticulosas

3. Nose

Recurrent epistaxis

4. Renal tract

a) Haemoturia

b) Hemoglobinuria

5. Lungs

a) Haemoptysis

II. Increased Requirements

Growing infants, children, Adolescents

Pre menopausal women

Menstruation

Pregnancy and lactation

III. Inadequate Dietary Intake

Poor economic status

Anorexia in pregnancy

Elderly individuals due to poor dentition, apathy and financial constraints

IV. Decreased Absorption

Partial or total gastrectomy

Achlorhydria

Intestinal malabsorption such as coelic disease pica

Females in reproductive period

The highest incidence of IDA is in women during their reproductive period it may be from the following causes

1.Blood loss:

Persistant and heavy menstrual blood has due to insertion of IUCD's may due to repeated miscarriage

2. Inadequate intake

3. Increased Requirements – during parturition and lactation.

Post menopausal females

Due to carcinoma of the uterus, bleeding from alimentary tract such as carcinoma of stomach etc,.

Adult males – very rare

Infants and children's -IDA is fairly common during 1-2 years of age

Hook Worm Infestation (Ankylostomiasis):

It is the important cause of intestinal blood loss. Parasite ancylostoma duodenale and nector americanus attaches to the proximal portion of the small intestine and suck blood from sub mucosal vessels. The amount of blood lost is a function of hookworm load,which turns in proportional to the number of ova in the stool each worm which has been in the intestine for months or years draws 0.2-0.5 ml of blood/ day. It has been estimated that the loss of hemoglobin for every twelve worms may be one percent feacal ova counts in excess of 500 g are associated with iron losses of more than 3 to 4 mg/day and a high incidence of IDA.

Regulation of iron in the body

When the body has become saturated with iron the rate of absorption of iron from intestinal tract becomes greatly decreased whereas if the iron stores is depleted of iron the rate of absorption is accelerated to 5 or more times than the normal rate of absorption.

Clinical features

*** General**

Weakness, fatigue, lassitude, swelling of the body and oedema, pallor, dry skin, lusterless hair, white sclera, spoon shaped deformity of nails(koilonychias)

*** Cardiovascular System**

Palpitation, breathlessness, angina pain, sinus tachycardia, collapsing pulse, dancing carotids, full neck veins, dilated heart with slightly shifted apex, functional systolic murmur over pulmonary and mitral area (haemic murmur), venous hum, congestive cardiac failure.

*** GI tract**

Anorexia, acidity, heartburn, palpable spleen and liver, clay, ice cube or starch pica.

*** Neurological**

Dizziness, giddiness, tingling, numbness, insomnia, dimness of vision, forgetfulness, lack of concentration , loss of memory.

*** Reproductive system**

Amenorrhoea, Menorrhagia, abortion, infertility.

*** Renal system**

Slight proteinuria may be present in severe anemia.

*** Pyrexia –**mild pyrexia may occur with severe anemia but marked fever is due to either the causative disorder or to some complicating factor.

Clinical features due to epithelial tissue changes

This is seen in few individuals having long standing iron deficiency anemia particularly in middle-aged women.

*** Tongue**

Atrophic glossitis resulting in bald tongue with soreness sometimes leukoplakia.

*** Mouth** – angular stomatitis with glossitis.

*** Oesophagus**

Obstruction of the oesophagus from spasm or rarely by a band or web consisting of denuded, aggregated epithelial cells may be seen in X-ray as "post cricoids web"

*** Nail -** Brittle, thinned out, flattened and sometimes spoon shaped nail.

*** Hair -** Thin and lustreles.

The association of chronic iron deficiency anemia with koilonchyia, glossitis, dysphagis and splenomegaly is called Plummer-Vinson syndrome or Paterson-kelley syndrome.

Complication of IDA

* IDA may be the present finding in gastrointestinal carcinoma

* In patients with heart disease, severe anemia may precipitate angina pectoris or congestive heart failure.

* Infections are more common in IDA especially those of the respiratory, gastrointestinal and urinary tracts.
* Chronic IDA reduces the efficacy in work and study.

Differential Diagnosis

In a patient with hypochromic microcytic anemia the major diagnostic possibilities are

i) Iron deficiency

ii) Thalassemia

iii) Anemia of chronic infection

iv) Sideroblastic anemia

Several laboratoty test are useful in the different at diagnosis.

i) Thalassemia

It mild forms of thalassemia microcytosis is much more marked than hypochromic accordingly the MCHC is usually normal the red cell distribution is more uniform than that in iron deficiency. Target cella and basophilic stippling are usually more prominent in thalassemia than iron deficiency, the serum iron is normal or elevated.

ii) Anemia of chronic infection

The serum iron is decreased the transferring level is also decreased the laboratory tests are not very helpful in determining whether a patient with chronic inflammatory disease such as rheumatoid arthiritis has become iron deficiency.

iii) Sideroblastic anemia

The diagnosis of sideroblastic anemia rests on the demonstration o ringed sideroblasts in the marrow these patient often have a population of hypochromic microcytic red cells eventhough red in cell indexes are usually normal.

Investigations:

I) Blood Investigation

1. Total red blood cell count
2. Differential count
3. Erythrocyte sedimentation rate
4. Mean corpuscular hemoglobin
5. Mean corpuscular volume
6. Mean corpuscular hemoglobin concentration
7. Packed cell volume
8. Red cell survival
9. Serum iron

10. Serum ferritin concentration
11. Serum protein
12. Serum creatinine

II) Urine investigation

1. Urine sugar
2. Urine albumin
3. Deposits

Red blood cells

Pus cells

III) Stools Investigation

1. Occult blood
2. Ova
3. Cyst
4. Red blood cells
5. Pus cells

From the above investigation the following are predominant in iron deficiency anemia

* In IDA the hemoglobin may fall to as low as 3gm/100ml but the red cell count is rarely below 2.5 million/cubic millimeter.
* The red cells are usually microcytic and hypochromic.

* Reticulocyte and platlets are normal or increased.
* The white cell count is normal serum iron is usually below 30 mug/100 ml (normal is 90-150 mug/100 ml)
* Bone marrow hemosidering is absent
* The MCHC is below 27gm/dl.

Diagnostic Features of Iron Deficiency Anemia:

Following criteria are essential to diagnose iron deficiency anemia

* History of inadequate intake of dietary iron and blood loss if any
* Typical symptoms and signs like easy fatigubility, waxy pallor, pica, koilonchyia, smooth tongue, dysphagia associated with general consideration
* Erythrocyte count may be normal or reduced less than 3 million/cumm
* Reduction in hemoglobin below 10 mg%
* Hypochromic and microcytic morphology of peripheral smear
* Low serum iron, increased total iron binding capacity
* Serum ferittin level is reduced
* Reduced mean cell volume
* Platlet count is either normal or raised
* Bone marrow haemociderin absent
* Occult blood present in stools

Management

This can be considered as follows

1. Correction of anemic state – overall correction of nutrition with articles rich in iron is important, iron deficiency is corrected by intake of rich iron content diet and administration of medicinal iron,

2. Replacement of iron stores

3. Elimination of the causes

DIET

Haem iron sources

Muscle meat

Organ meat (e.g.liver)

Fish and shell fish

Non Haem iron sources - Oatmeal, legume (peas, beans) dried nuts, whole meal, green leafy vegetables, fortified iron salt, cereal foods, food rich in vitamin c in the same meal enhances iron absorption.

Prophylaxis

There are three main principles in the prevention of nutritional iron deficiency anemia.

1. The regular consumption of a well balanced diet containing an adequate quantity of iron.

2. The periodic administration of iron as drug during increased physiological demands of women e.g. pregnancy, lactation and menstruation.

3. Maintenance of a normal hemoglobin level in the mother is desirable for the prevention of iron deficiency anemia infants, premature and unduly small infants should be given prophylactic iron as a routine therapy, iron rich sources should be added in the infants diet from the third or fourth month and thereafter be progressively increased. Following control of infection, iron should be given to all infants and children's if the hemoglobin level is reduced.

MATERIALS AND METHODS

An open labelled randomised clinical trial on **"Vatha Pandu"** was conducted in Government Siddha Medical College and Hospital, Palayamkottai, Tirunelveli. Totally fourty cases were selected 20 patients treated as OP remaining 20 patients were treated as IP. The clinical signs and symptoms of **"Vatha Pandu"** of both sexes of different ages were selected and studied under the guidance of the professor, reader and lecturer of P.G. Pothu Maruthuvam Department.

Preparation of Trial Medicine

The trial medicine **'Karisalankanni chooranam'** is selected based on their medicinal values in treating **'Vatha Pandu'** as mentioned in the siddha literature **Sigitcharathnadeepam – part II** (page no.162). The ingredients are collected and purified and the medicine were prepared, stored and dispensed in pockets containing 2gms each to the patients based on the reference cited above.

Parameters for case selection

Cases were selected from outpatient and inpatient department.

Inclusion criteria

The parameters for the selection were insidious

Onset of easy fatigubility

Weakness

Headache

Bodyache

Inability to concentrate

Giddiness

Pallor of skin

Conjunctiva

Mucous membrane of lips and pale tongue

Koilonychia

Hb level between 7.1-10 gm%

Worm infestations.

Exclusion criteria

Patients are excluded in the following conditions

Hb less than 7

Congenital heart disease

Chronic renal diseases

History of liver diseases

Inherited defects

Haemorrahgic disorders

Thalassemia

Ischemic heart disease

Thyroid disorder

Diabetes mellitus.

Withdrawal criteria

Patient are advised to withdraw in following conditions Intolerance to the drug and development of any serious adverse effects during the trial, Patients turned unwilling to continue in the course of clinical trial, Poor compliance, Any other acute illness which need rescue medication and to follow regular diet schedule.

Clinical examination

Patients were subjected to physical examination on siddha methodology **"Piniyari Muraimai"**. It has three main principles which are Poriyal Arithal, Pulanal Arithal and Vinathal and effected through **'Envagai Thervugal'**. Coming to the modern methodology detailed clinical history was recorded. All the patients are subjected to the following investigations

Siddha Assesment

- Neerkuri – Physical Urine examination.
- Neikuri – Oil examination
- Naadi paritchai(including Envagaithervugal) – Pulse examination

Investigations

Blood - Complete blood analysis TC, DC, Hb, TRBC, ESR, PCV. MCV, MCH, MCHC, Peripheral blood smear were carried out.

Routine Urine Analysis- Albumin, Sugar, Deposits, Bile salts, Bile pigment are noted.

Stools- for Ova, Cyst and Occult blood were also done.

Biochemical Analysis- Serum Cholesterol, Serum Protein, Serum Bilirubin, Serum Ferritin, TIBC, Blood Sugar, Blood urea are carried out before the treatment and at the time of discharge.

Methods of Treatment

Siddha system of medicine is based on mukkutra theory and hence the treatment is mainly aimed to bring down the three dhoshas to its equilibrium state and thereby restoring the physiological condition of three dhoshas.

The clinical improvement was followed and it was recorded in the case sheet.

Bio-chemical and Pharmacological studies of the drug were done to make out the active principles and action of the trial medicine.

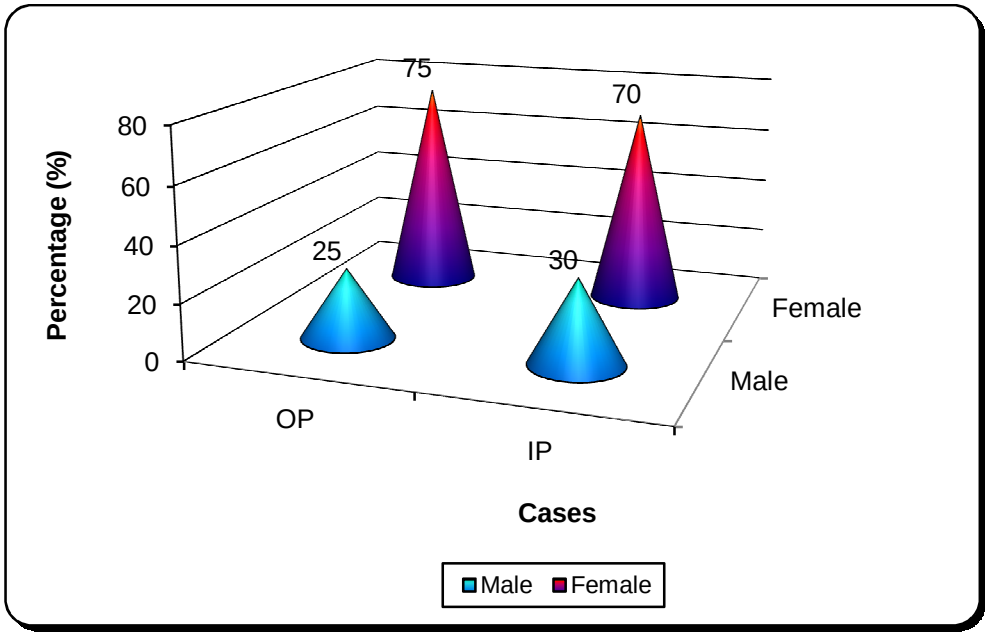
OBSERVATION AND RESULTS

The results were observed on the basis of the following criteria by conducting clinical study on 20 inpatients and 20 outpatients totally 40 patients.

- Sex Distribution
- Age Distribution
- Kaalam
- Paruvakaalam
- Thinai
- Occupational status
- Socio economic status
- Food habits
- Personal habits
- Aetiological factors
- Mukkutram- Vatham, Pitham, Kabam
- Ezhu Udal Thathukkal
- Enn vagai Thervugal
- Neerkuri , Neikuri
- Clinical Features and prognosis
- Haemoglobin level
- Assessment of results by Laboratory investigation.

1.SEX DISTRIBUTION

Sl.No	Sex	No. of Cases		Percentage (%)	
		OP	IP	OP	IP
1.	Male	5	6	25	30
2.	Female	15	14	75	70

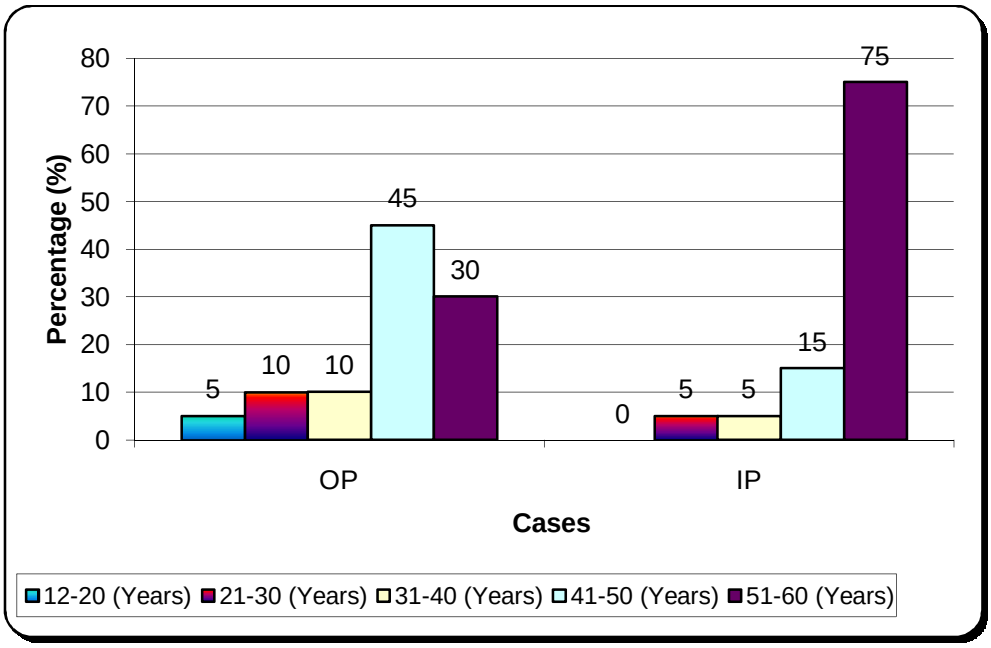


Inference :

Among 40 cases, 29 Patients were females and 11 Patients were males.

2.AGE DISTRIBUTION

Sl.No	Age in Years	No. of Cases		Percentage (%)	
		OP	IP	OP	IP
1.	12-20 (Years)	1	-	5	-
2.	21-30 (Years)	2	1	10	5
3.	31-40 (Years)	2	1	10	5
4.	41-50 (Years)	9	3	45	15
5.	51-60 (Years)	6	15	30	75

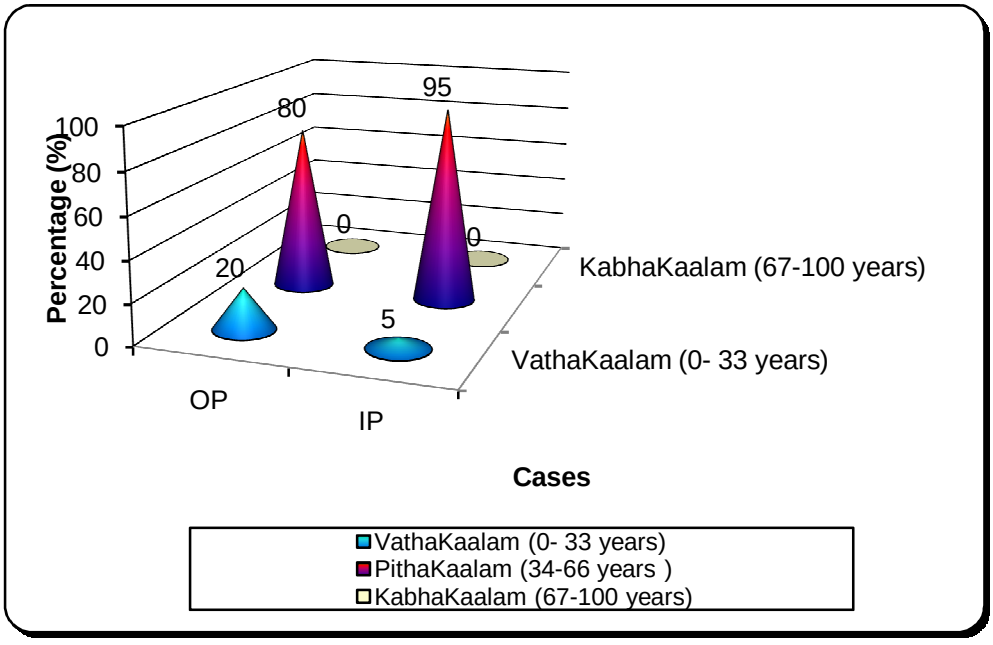


Inference:

Out of 40 patients, 21 Patients were in the age group of 51-60, 12 Patients were in the age group of 41-50, 3 Patients were in the age group of 31- 40, 3 Patients were in the age group of 21-30, 1 Patient were in the age group of 12-20.

3. DISTRIBUTION OF KAALAM

Sl.No	Kalam	No. of Cases		Percentage (%)	
		OP	IP	OP	IP
1.	VathaKaalam 0- 33 years	4	1	20	5
2.	PithaKaalam 34-66 years	16	19	80	95
3.	KabhaKaalam 67-100 years	-	-	-	-

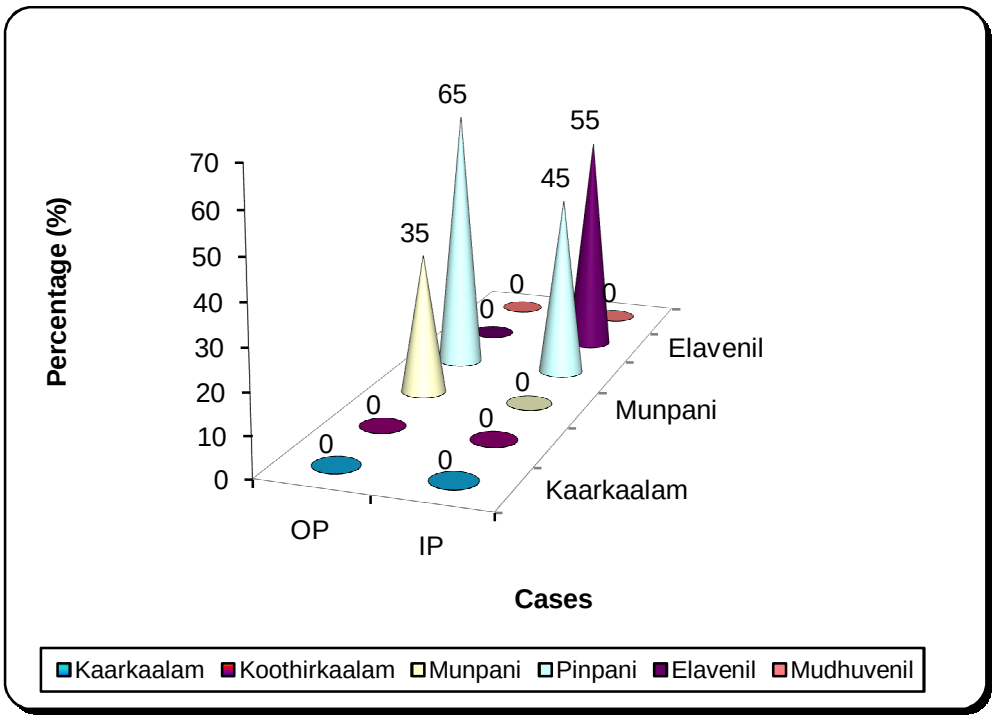


Inference:

Out of 40 patients, 5 Patients comes under VathaKaalam and 35 Patients comes under PithaKaalam.

4.PARUVAKAALAM DISTRIBUTION:

Sl.No	Paruva Kaalam	Months	No. of Cases		Percentage (%)	
			OP	IP	OP	IP
1.	Kaarkaalam	Avani, Puratasi, Mid Aug-Mid Oct	**0**	**0**	**0**	**0**
2.	Koothirkaalam	Iyppasi, Kaarthigai Mid Oct-Mid Dec	**0**	**0**	**0**	**0**
3.	Munpani	Margazhi, Thai Mid Dec-Mid Feb	**7**	**0**	**35**	**0**
4.	Pinpani	Maasi, Panguni Mid Feb-Mid April	**13**	**9**	**65**	**45**
5.	Elavenil	Chithirai, vaigasi Mid April- Mid June	**0**	**11**	**0**	**55**
6.	Mudhuvenil	Aani, Aadi Mid June-Mid Aug	**0**	**0**	**0**	**0**

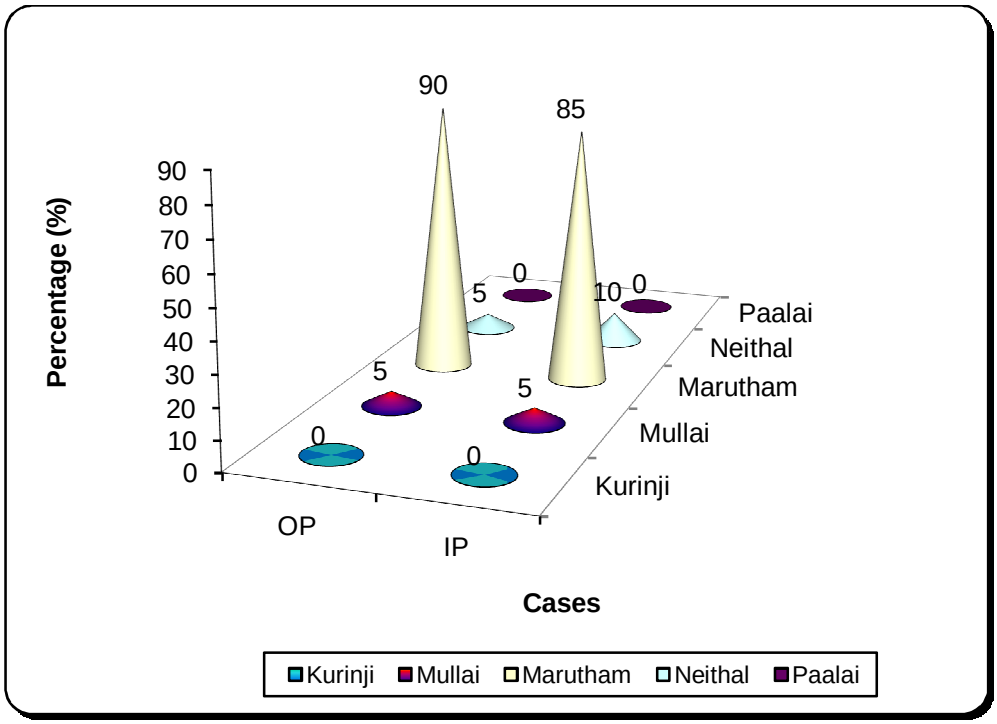


Inference:

Out of 40 patients, 22 Patients comes under Pinpani , 11 patients comes under Elavenil, 7 Patients comes under Munpani kaalam.

5.DISTRIBUTION OF THINAI

Sl.No	Distribution	No. of Cases		Percentage (%)	
		OP	IP	OP	IP
1.	Kurinji	0	0	0	0
2.	Mullai	1	1	5	5
3.	Marutham	18	17	90	85
4.	Neithal	1	2	5	10
5.	Paalai	0	0	0	0

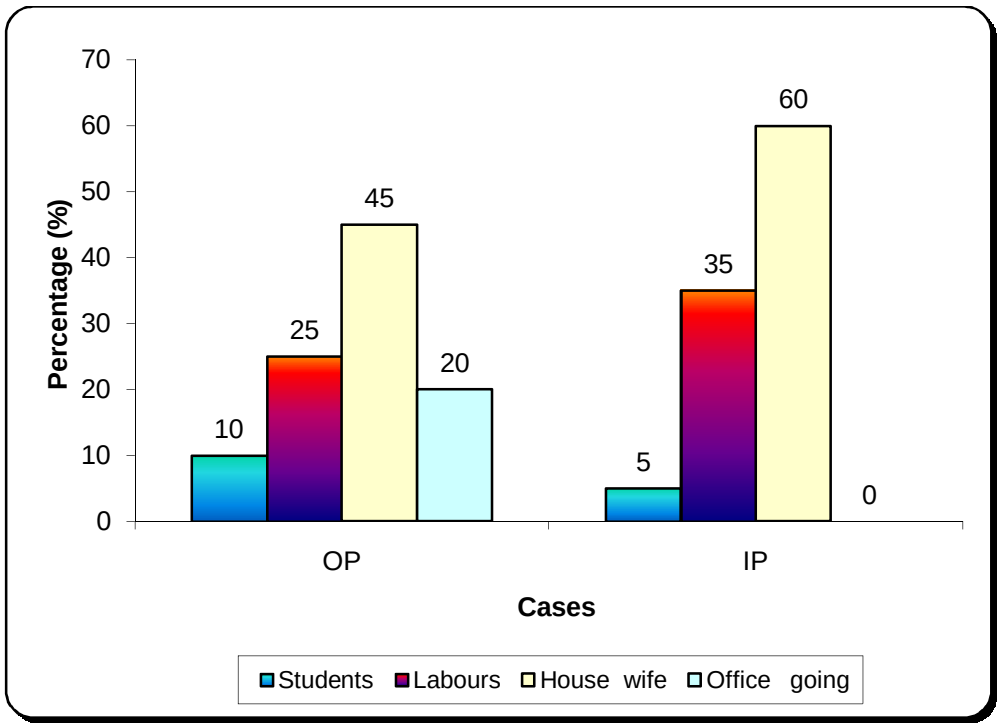


Inference:

Out of 40 patients, 35 Patients comes under Neithal and 5 patients comes underMarutham.

6. OCCUPATIONAL STATUS

Sl.No	Occupational Status	No. of Cases		Percentage (%)	
		OP	IP	OP	IP
1.	Students	2	1	10	5
2.	Labours	5	7	25	35
3.	House wife	9	12	45	60
4.	Office going	4	0	20	0

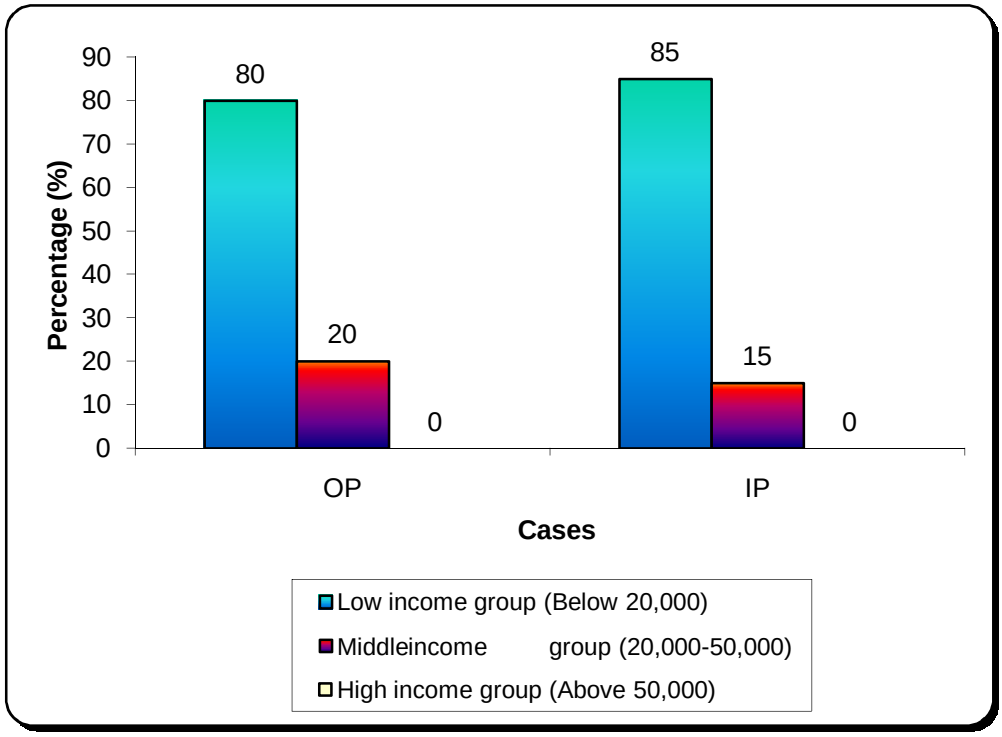


Inference:

Out of 40 patients, 21 Patients were House wife, 12 Patients were Labours, 4 were office going and remaining 3 Patients are students.

7. SOCIO-ECONOMIC STATUS

Sl.No	Income / Month	No. of Cases		Percentage (%)	
		OP	IP	OP	IP
1.	Low income group (Below 20,000)	16	17	80	85
2.	Middleincome group (20,000-50,000)	4	3	20	15
3.	High income group (Above 50,000)	0	0	0	0

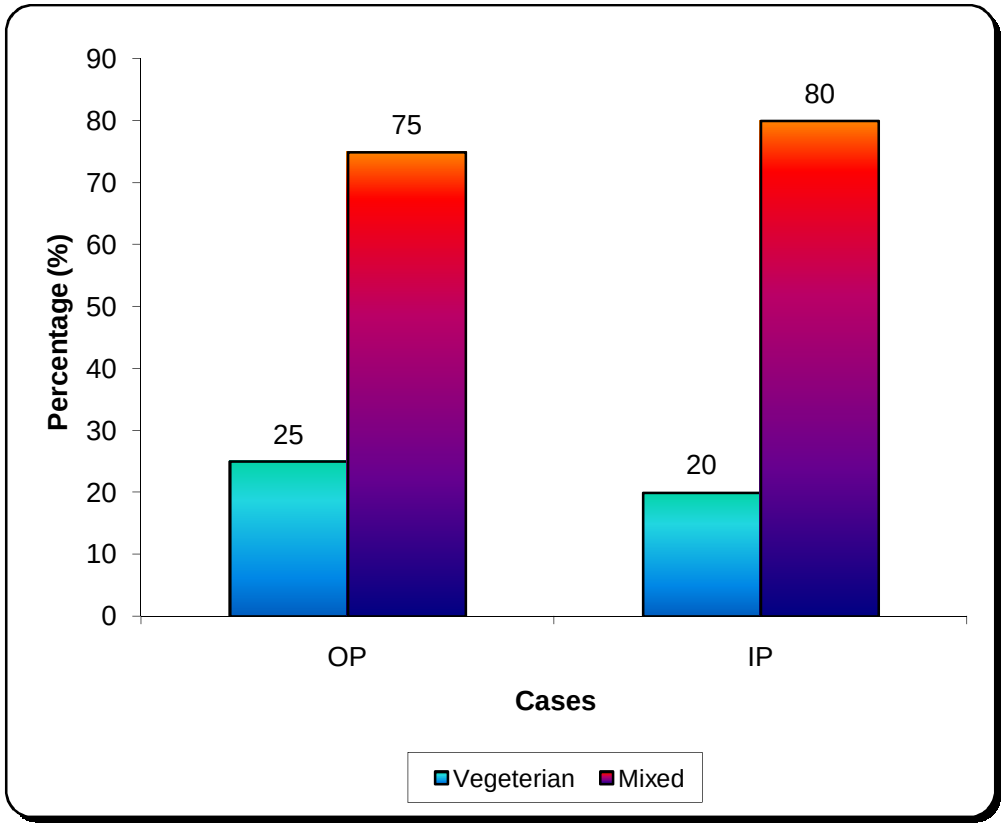


Inference:

Out of 40 patients, 33 Patients belongs to low income group , 7 Patients belongs to middle income group.

8.DIETARY HABITS

Sl.No	Dietary Habits	No. of Cases		Percentage (%)	
		OP	IP	OP	IP
1.	Vegeterian	5	4	25	20
2.	Mixed	15	16	75	80

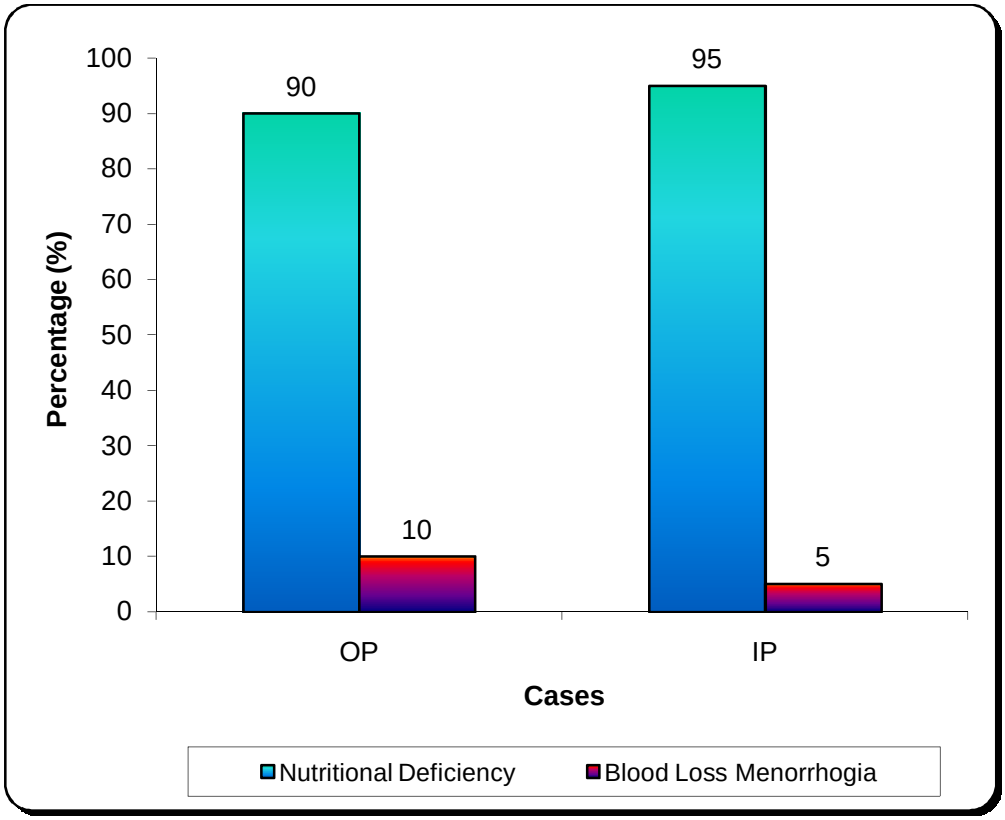


Inference:

Out of 40 patients, 31 Patients were mixed diet and 9 Patients were vegetarian.

9. AETIOLOGICAL FACTORS

Sl.No	Aetiology	No. of Cases		Percentage (%)	
		OP	IP	OP	IP
1.	Nutritional Deficiency	18	19	90	95
2.	**Blood Loss** Menorrhagia	2	1	10	5
3.	Piles	0	0	0	0

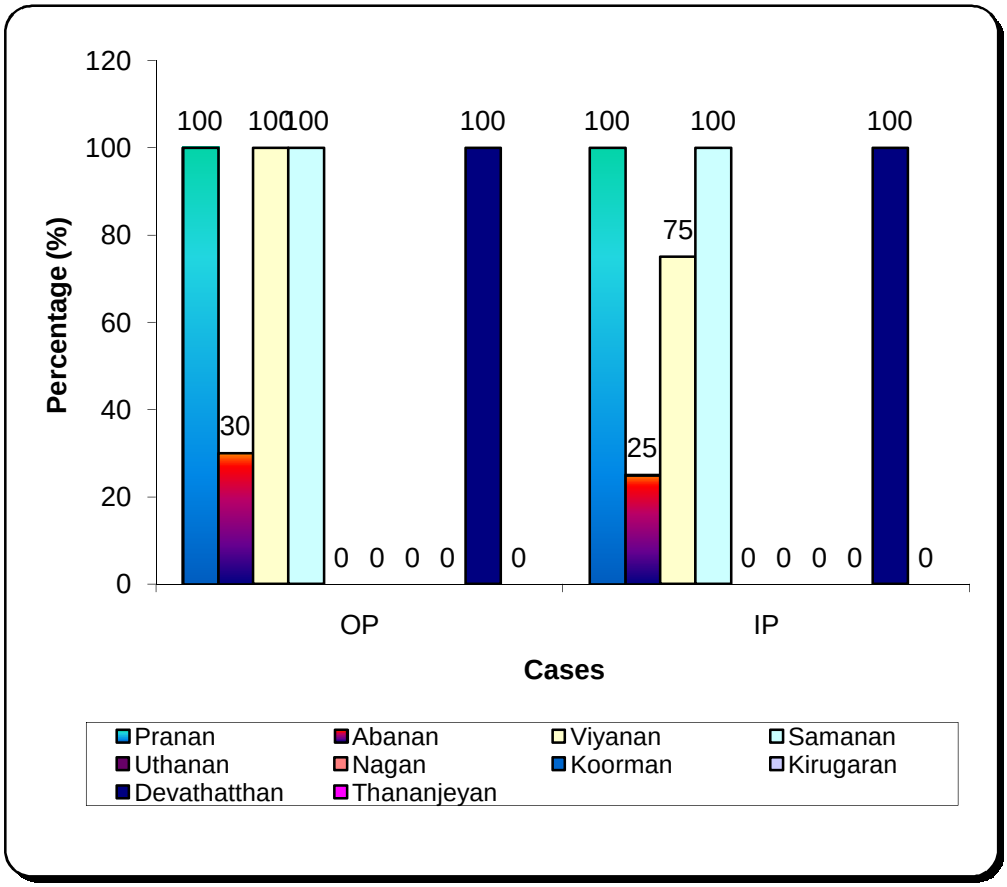


Inference:

Out of 40 patients, 37 patients were due to nutritional deficiency.

10.DISTRIBUTION OF VATHAM

Sl.No	Vatham	No. of Cases		Percentage (%)	
		OP	IP	OP	IP
1.	Pranan	20	20	100	100
2.	Abanan	6	5	30	25
3.	Viyanan	20	15	100	75
4.	Samanan	20	20	100	100
5.	Uthanan	0	0	0	0
6.	Nagan	0	0	0	0
7.	Koorman	0	0	0	0
8.	Kirugaran	0	0	0	0
9.	Devathatthan	20	20	100	100
10.	Thananjeyan	0	0	0	0

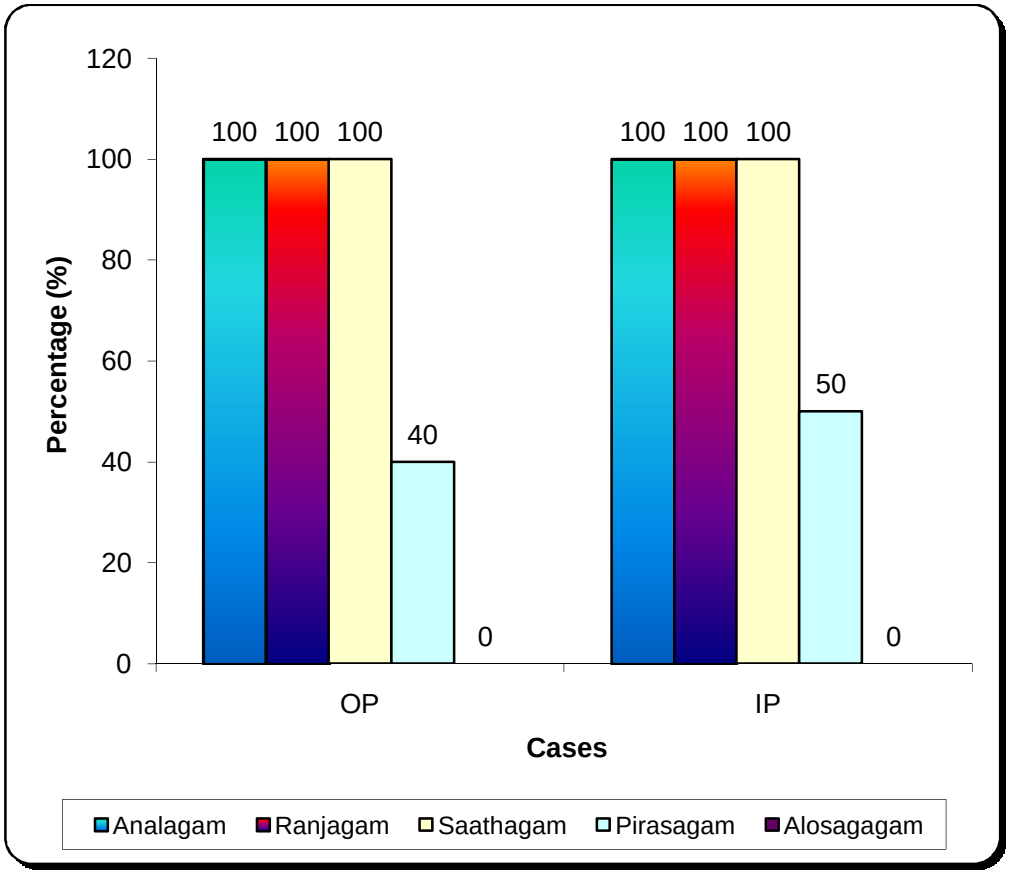


Inference:

Pranan, samanan, devathathan were affected in all patients, Abanan affected in 11 patients, Vyanan affected in 35 patients.

11.DISTRIBUTION OF PITHAM

Sl.No	Pitham	No. of Cases		Percentage (%)	
		OP	IP	OP	IP
1.	Analagam	20	20	100	100
2.	Ranjagam	20	20	100	100
3.	Saathagam	20	20	100	100
4.	Pirasagam	8	10	40	50
5.	Alosagagam	0	0	0	0

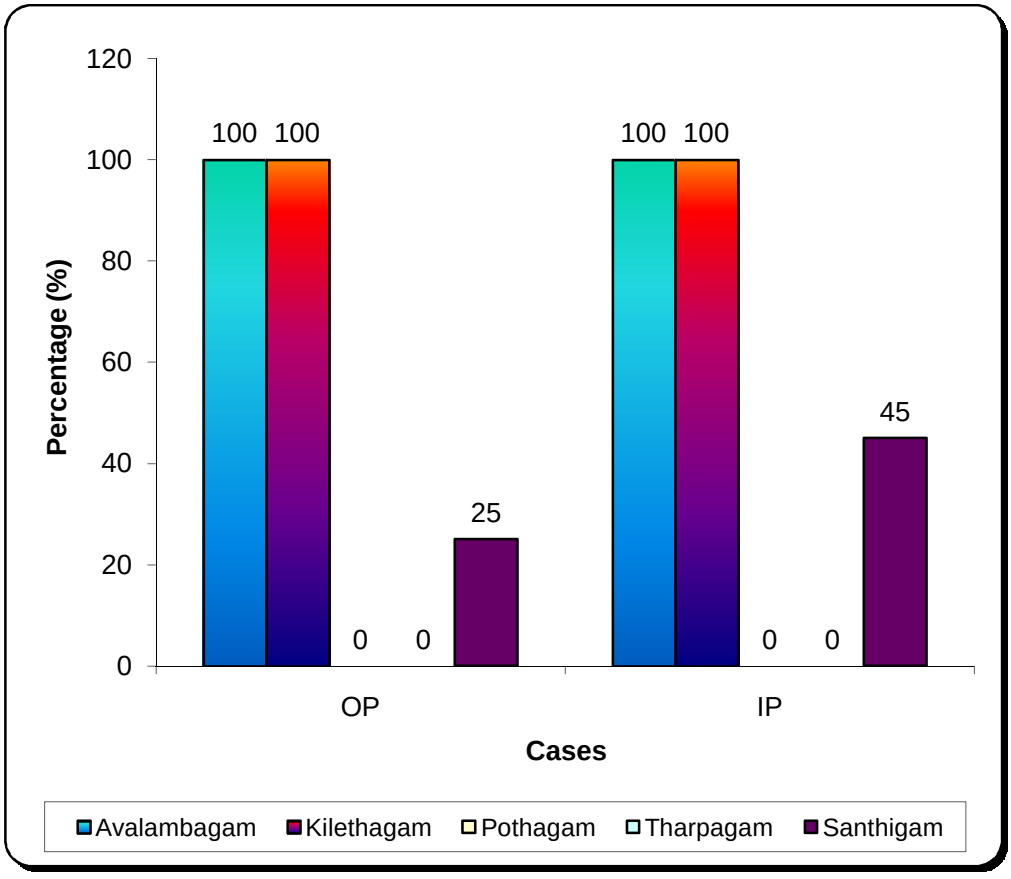


Inference:

Out of 40 patients, Analagam, Ranjagam, Sathagam were affected in all patients, Pirasagam were affected in 18 patients.

12.DISTRIBUTION OF KABAM

Sl.No	Kabam	No. of Cases		Percentage (%)	
		OP	IP	OP	IP
1.	Avalambagam	20	20	100	100
2.	Kilethagam	20	20	100	100
3.	Pothagam	0	0	0	0
4.	Tharpagam	0	0	0	0
5.	Santhigam	5	9	25	45

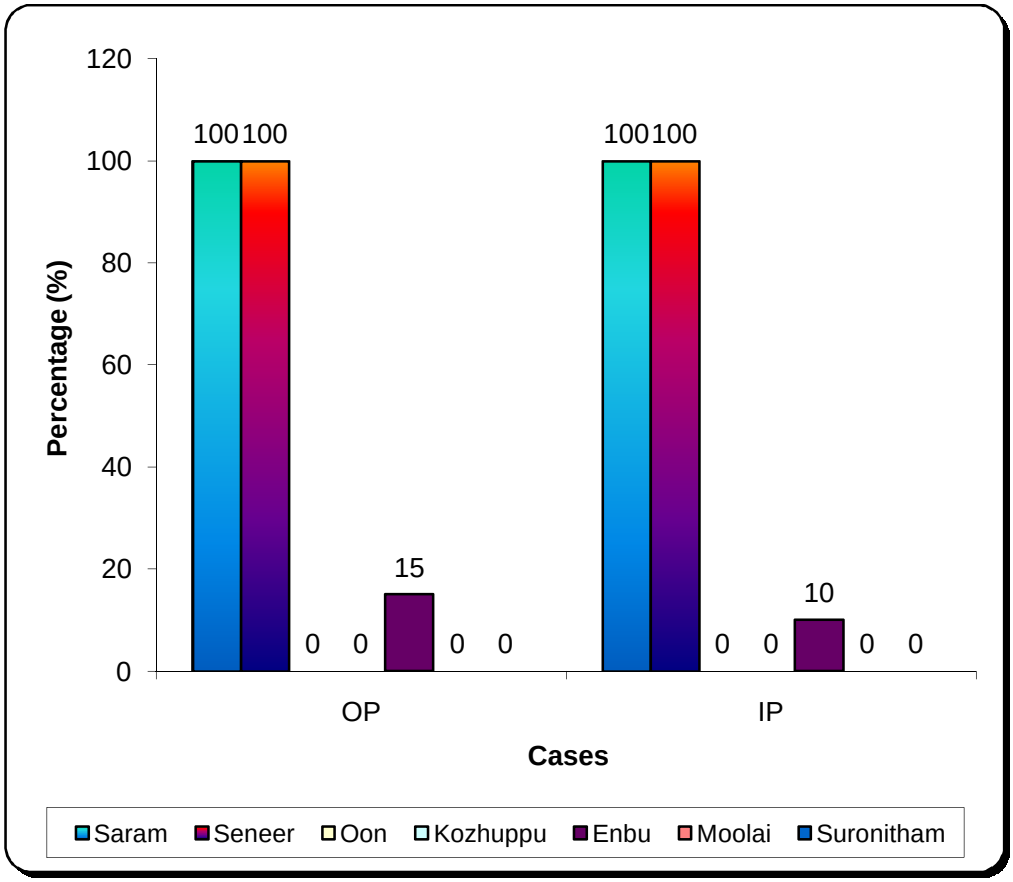


Inference:

Out of 40 patients, Avalambagam and Kilethagam affected in all patients, Santhigam affected in 14 patients.

13.EZHU UDAL THATHUKKAL

Sl.No	Ezhu Udal Thathukkal	No. of Cases		Percentage (%)	
		OP	IP	OP	IP
1.	Saram	20	20	100	100
2.	Seneer	20	20	100	100
3.	Oon	0	0	0	0
4.	Kozhuppu	0	0	0	0
5.	Enbu	3	2	15	10
6.	Moolai	0	0	0	0
7.	Suronitham	0	0	0	0

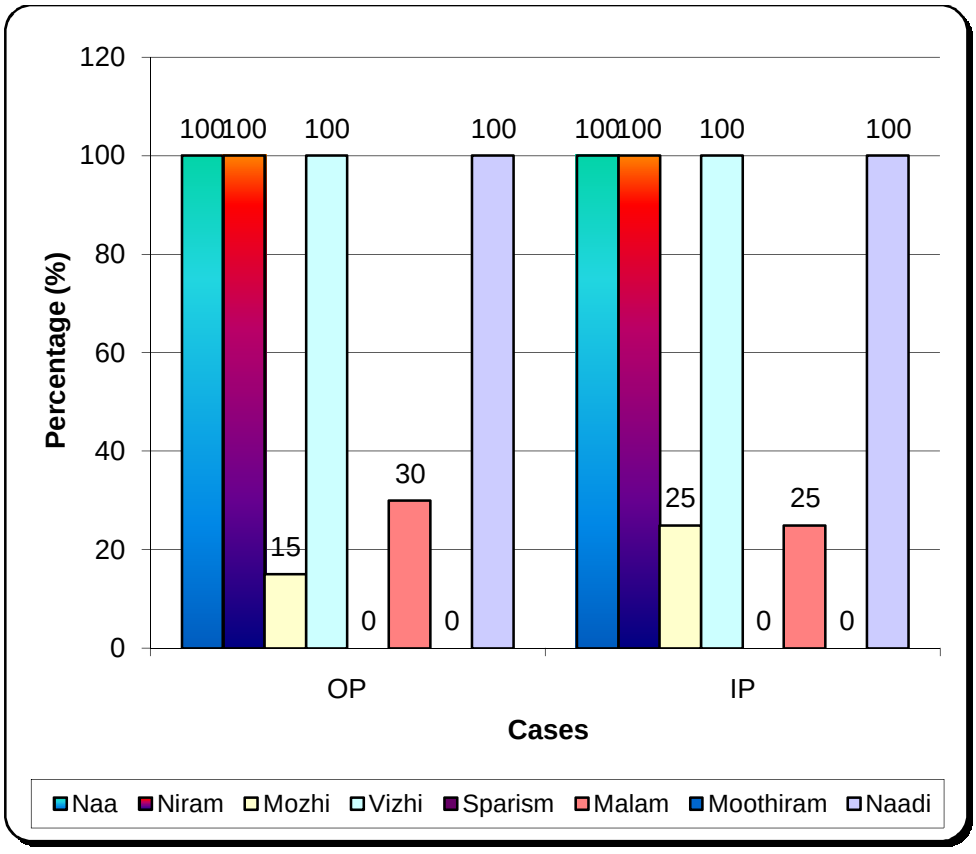


Inference:

Out of 40 patients, Saaram and senneer affected in all patients.

14.ENN VAGAI THERVUGAL

Sl.No	Envagai Thervugal	No. of Cases		Percentage (%)	
		OP	IP	OP	IP
1.	Naadi	20	20	100	100
2.	Sparism	0	0	0	0
3.	Naa	20	20	100	100
4.	Niram	20	20	100	100
5.	Mozhi	6	10	15	25
6.	Vizhi	20	20	100	100
7.	Malam	6	5	30	25
8.	Moothiram	0	0	0	0

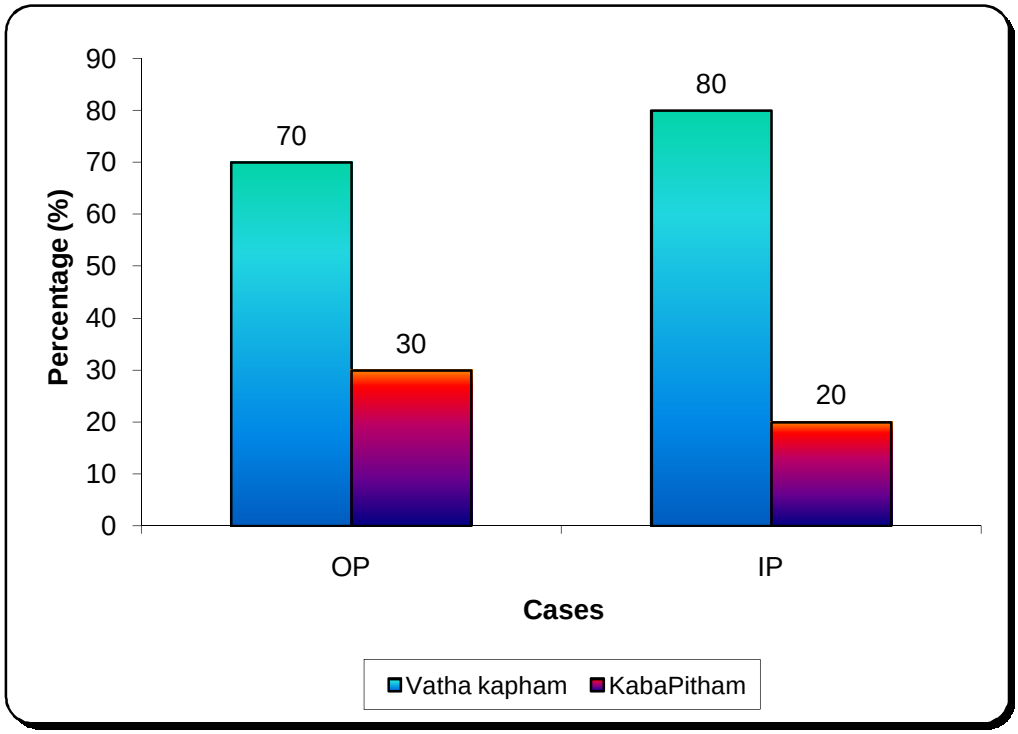


Inference:

Naa, Niram ,Vizhi and Naadi were affected all patients, Malam affected in 11 patients.

15.NAADI

Sl.No	Naadi	No. of Cases		Percentage (%)	
		OP	IP	OP	IP
1.	Vathakabam	14	16	70	80
2.	KabhaPitham	6	4	30	20

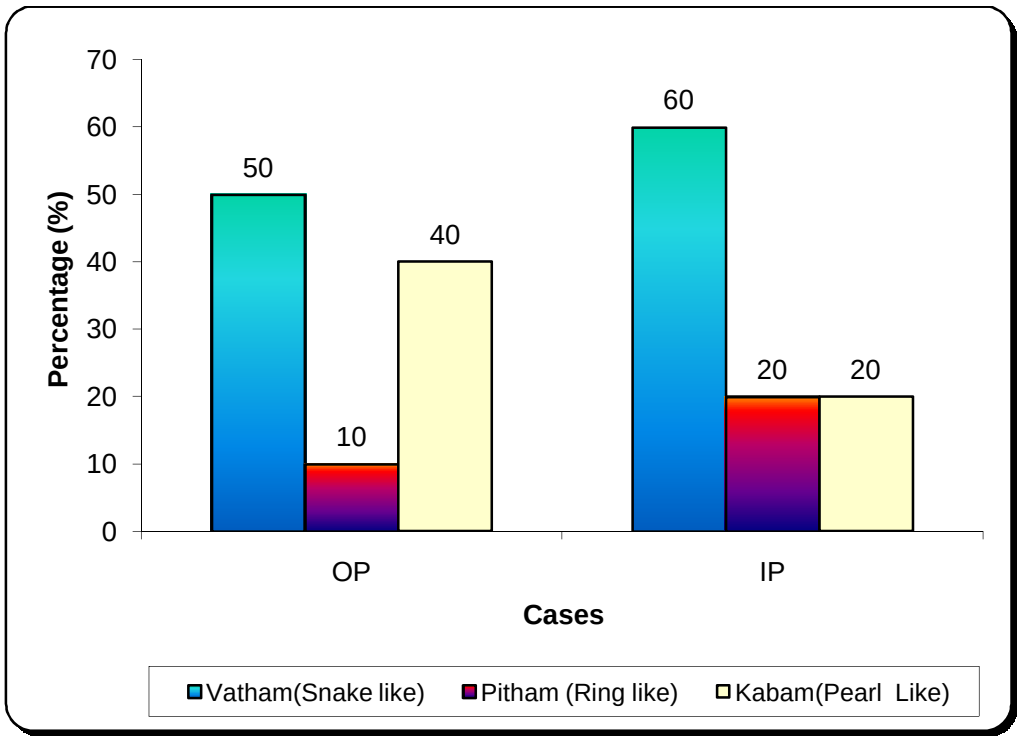


Inference:

30 patients had Vathakaba Naadi, and 10 patients had Kabhapitha Naadi .

16. NEIKURI

Sl.No	Neikuri	No. of Cases		Percentage (%)	
		OP	IP	OP	IP
1.	Vatham(Snake like)	10	12	50	60
2.	Pitham (Ring like)	2	4	10	20
3.	Kabham(Pearl Like)	8	4	40	20

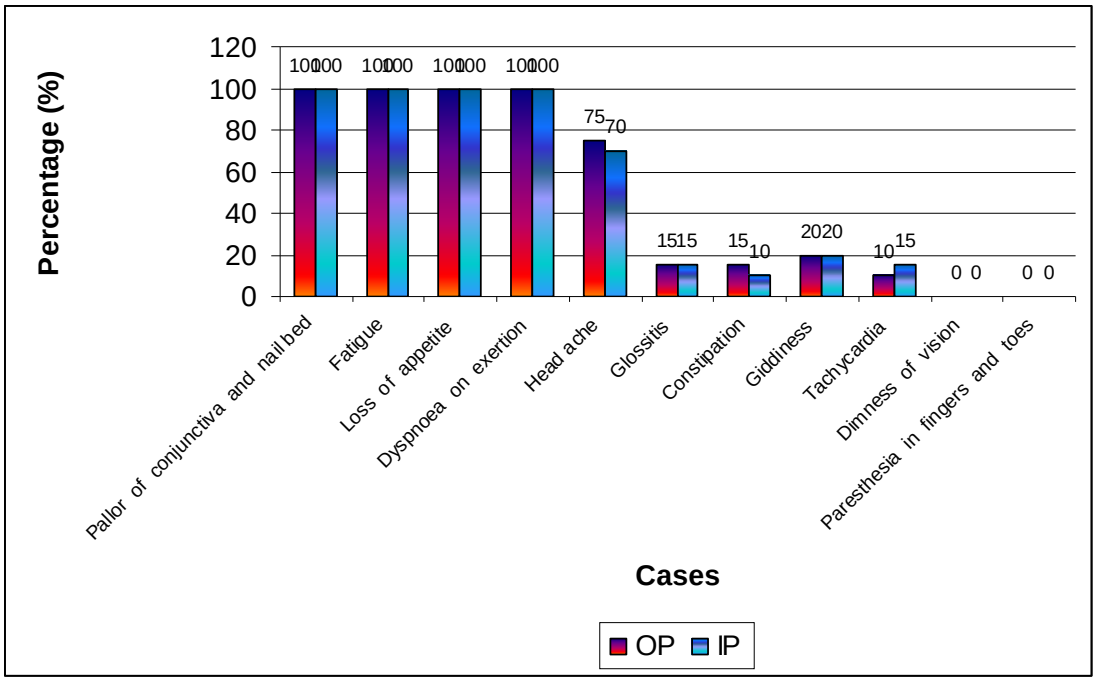


Inference:

Out of 40 patients, 22 patients had Vatha Neer, 12 patients had Kabha Neer and 6 patients had Pitha Neer Neikuri.

17. CLINICAL FEATURES

Sl.No	Clincal Features	No. of Cases		Percentage (%)	
		OP	IP	OP	IP
1.	Pallor of conjunctiva and nail bed	20	20	100	100
2.	Fatigue	20	20	100	100
3.	Loss of appetite	20	20	100	100
4.	Dyspnoea on exertion	20	20	100	100
5.	Head ache	15	14	75	70
6.	Glossitis	3	3	15	15
7.	Constipation	6	5	30	25
8.	Giddiness	10	8	50	40
9.	Tachycardia	2	3	10	15
10.	Dimnished of vision	0	0	0	0



Inference:

Out of 40 patients, 40 patients had Pallor of conjunctiva and nail bed, Fatigue, Loss of appetite, Dyspnoea on exertion, 29 Patients had Headache, 6 patients had Glossitis, 11 patients had Constipation, 18 patients had Giddiness and Tachycardia.

18. CLINICAL PROGNOSIS

Sl.No	Signs & Symptoms	Before Treatment				After Treatment			
		No. of Cases		Percentage (%)		No. of Cases		Percentage (%)	
		OP	IP	OP	IP	OP	IP	OP	IP
1.	Pallor of conjunctiva and nail bed	20	20	100	100	1	1	5	5
2.	Fatigueness	20	20	100	100	1	1	5	5
3.	Loss of appetite	20	20	100	100	1	1	5	5
4.	Dyspnoea on exertion	20	20	100	100	3	2	15	10
5.	Headache	15	14	75	70	1	3	5	15
6.	Glossitis	3	13	15	15	1	0	5	0
7.	Constipation	6	5	30	25	0	0	0	0
8.	Giddiness	10	8	50	40	0	0	0	0
9.	Tachycardia	4	4	20	20	0	0	0	0

Inference:

After treatment Pallor of conjunctiva and nail bed , Loss of appetite and Fatigueness present in 2 patients, Dyspnoea on exertion present in 5 patients, Headache present in 4 patients, Glossitis present in 1 patient.

19.HAEMOGLOBIN LEVEL OF OUT PATIENTS

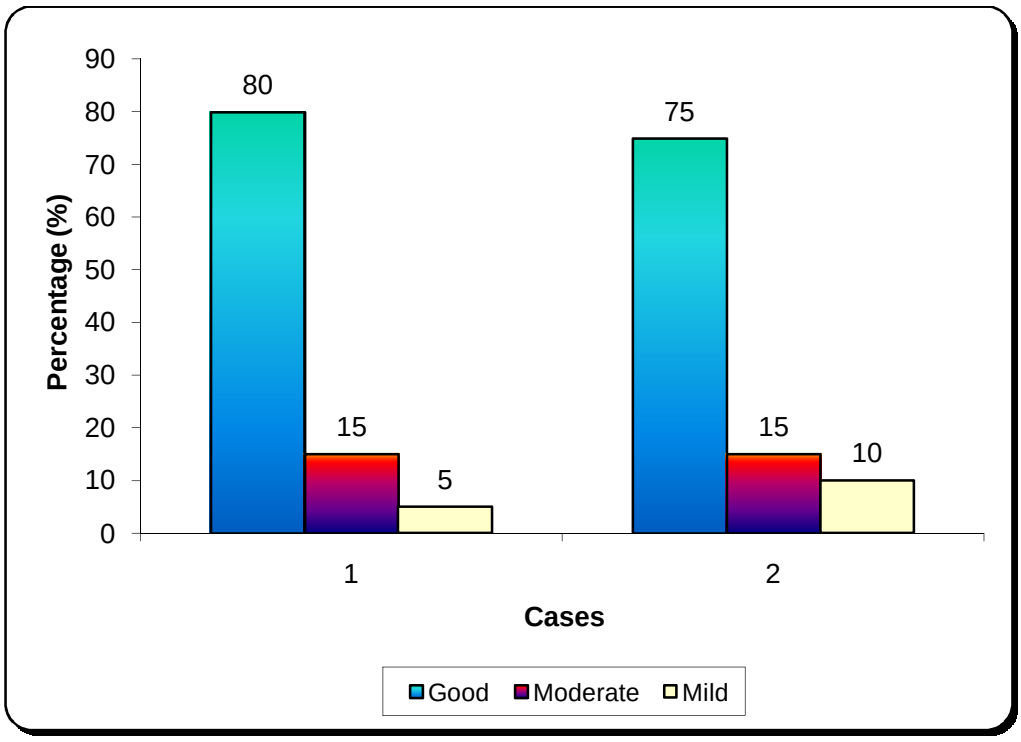
S. No	O.P. No	NAME	BeforeTreatment (gms/dl)	AfterTreatment (gms/dl)
1.	12963	Kalaivani	9.4	12.4
2.	12866	Janani	8.3	11.5
3.	13206	Sivaranjani	9.6	12.6
4.	13962	Shanthi	9.2	11.5
5.	13963	Pappathi	9.1	12.5
6.	14074	Kalyani	8.8	12
7.	15376	Rajeshwari	7.8	11.6
8.	15622	Maheswari	7.1	10.2
9.	16301	Mary	7	10.5
10.	17775	Geetha	7.7	10.4
11.	18972	Murugan	9.4	12.7
12.	19107	Anantham	9.8	11.8
13.	19538	Chelladurachi	9.9	12.4
14.	19597	Rekha	7	8.2
15.	24459	Sundari	8.3	11.5
16.	20311	Kowdhulalam	9.4	12.6
17.	20371	Jesuraj	9.8	11.5
18.	21436	Leela	9.5	12.5
19.	21913	Ganesan	8.5	12
20.	21914	Subramanium	8	11.6

20. HAEMOGLOBIN LEVEL OF IN PATIENTS

S. No	I.P. No	NAME	BeforeTreatment(gms/dl)	AfterTreatment(gms/dl)
21.	401	Pichammal	8	11.6
22.	478	Pappa	9.6	12
23.	571	Shanmugakani	9.2	12.2
24.	406	Jothi	9	12.1
25.	611	Selvabackiyam	9	12.5
26.	667	Subbu	8.4	11.5
27.	736	Thaiammal	8.6	11.6
28.	544	Gomathi	8.8	11.8
29.	954	Ponnukutti	8	10.2
30.	962	Sendu	9.3	11.5
31.	980	Pappa	8.5	11.1
32.	983	Gurusamy	8.8	11.8
33.	953	Muthu	9.8	12
34.	1000	Rathinammal	9.2	10.5
35.	1025	Varatharajan	7.6	8.7
36.	1070	Murugan	8.5	11.5
37.	1105	Mohammed	8.4	10.5
38.	1121	Subammal	7.2	10.5
39.	1148	Perumal	9.1	12.5
40.	964	Mookkammal	8.4	11.5

21. GRADING OF RESULTS

Sl.No	Grading of results	No. of Cases		Percentage (%)	
		OP	IP	OP	IP
1.	Good Response	16	15	80	75
2.	Moderate Response	3	3	15	15
3.	Poor Response	1	2	5	10



Good Response : Increase in Hb 3gm%and above after treatment

Moderate Response : Increase in Hb 2gm%after treatment

Poor Response: Increase in Hb 1gm% after treatment

Inference:

Out of 40 patients, 31 cases showed good result, 6 cases showed moderate result, 3 cases showed poor result.

ANNUXURE I

TRIAL DRUG

PREPARATION AND PROPERTIES OF TRIAL MEDICINE

KARISALANKANNI CHOORANAM

INGREDIENTS:

S.N	Name	Botanical Name	Parts used	Quantity
1.	Karisalankanni	*Eclipta prostrata*	Dried Leaves	4 parts
2.	Mookiratai	*Boerhaevia diffusa*	Dried Whole plant	1 part
3.	Chukku	*Zingiber officinale*	Dried Rhizome	1 part
4.	Milagu	*Piper nigrum*	Dried Seed	1 part
5.	Thippili	*Piper longum*	Dried Fruit	1 part
6.	Kadukkai	*Terminalia chebula*	Dried Thol	1 part
7.	Nellikkai	*Phyllanthus emblica*	Dried Fruit	1 part
8.	Thandrikai	*Terminalia bellerica*	Dried Fruit	1 part
9.	Maramanjal	*Coscinium fenestratum*	Dried Wood	1 part
10.	Thaniya	*Coriandrum sativum*	Dried Fruit	1 part
11.	Athimathuram	*Glycyrrhiza glabra*	Dried Root	1 part
12.	Karunseeragam	*Nigella sativa*	Dried Seed	1 part
13.	Thalisapathiri	*Abies spectabilis*	Dried Leaves	1 part
14.	Elam	*Elettaria cardamomum*	Dried Seed	1 part
15.	Seeragam	*Cuminum cyminum*	Dried Seed	1 part

METHOD OF PREPARATION:

The purified drugs are powdered separately and sieved in pure white cloth, then the powder of all the drugs are mixed and taken as a compound preparation.

DRUG DOSAGE: 2 gm , twice a day after food. With sugar.

INDICATION OF TRIAL MEDICINES: Vatha Paandu (Anaemia)

REFERENCE: *Sigitcharathnadeepam Part II,Page no 162.*

2.MOOKIRATAI

Common name	Í , Aó† ¬ ì
Botanical name	Boerhaavia diffusa
Family	Nyctaginaceae
Synonyms	¹ †ðè‹, Í , ° ø† ¬ ì, Þó^ î ¹ †Hè£
Part used	whole plant
Chemical constituents	Punaravine,sitosterol, aminoacid, fatty acids

Characters:

Suvai - kaippu

Thanmai - veppam

pirivu - karppu

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Action:

Expectorant, Diuretic, laxative, refrigerant, anthelmintic, emetic.

4.MILAGU

Common name	l ÷ °
Botanical name	Piper nigrum
Family	Piperaceae
Synonyms	è L ¬ ù , è P, «è£ ÷ è ‹ , ñ£ê ‹ , ê¼ñð‰ î ‹ , ñ ¬ ô ò£O Fó ƒ è™.
Part used	seed
Chemical constrituents	Chavicine , piperine , piperidine, pipertine, oleoresin.

Characters:

Suvai - kaippu, karppu

Thanmai - veppam

pirivu - karppu

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Action:

Carminative, Antiperiodic, Rubefacient, stimulant,Antivatha , Antidote.

6. KADUKKAI

Common name	è´ , è£Œ
Botanical name	Terminalia chebula
Family	combretaceae
Synonyms	Ü , «è£ì ‹ , Ü ƒ è í ‹ , Üðó í ‹ , Ü ¬ ðò ¡ , Ü ‹¼î£, ÜKî A, «êî A, Mü ò«õ î ¡ , õ ù ¶ ˜ , A
Part name	Thol
Chemical constituents	Tannin, gallicacid, chebulin, chebulinic acid.

Characters

Suvai - Thuvarppu

Thanmai - Veppam

Piravi - Inipu

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8 .THANDRIKAI

Common name	î£¡ P , è£Œ
Botanical name	Terminalia bellerica
Family	Combretaceae
Synonyms	Ü º î ‹, è‰îè†ðô ‹, õ£‰Fò ‹, Ì î õ£êè ‹, î£ E , è£Œ
Part used	fruit
Chemical constituents	Tannic acid

Characters:

Suvai - Thuvarppu

Thanmai - veppam

Pirivu - Inippu

Action:

Astringent,expectorant, laxative, tonic

10. THANIYA

Common name	î Q ò£, ª è£ˆ ¶ ñ™ L
Botanical name	Coriandrum sativum
Family	Apiaceae
Synonyms	à¼œ ÜKC
part used	Dried fruit
Chemical constituents	Fattyacid, volatile oil, flavanoides

Characters:

Suvai - karppu

Thanmai - seetha veppam

Pirivu - karppu

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Action:

Stomachic, carminative, stimulant,Diuretic

12. THALISAPATHRI

Common name	î£Oêðˆ FK
Botanical name	Abies spectabilis
Family	Taxaceae
Synonyms	î£Oêðˆð‹
Part used	Leaves
Chemical constituents	Taxol, Taxine ,Tannin,Ephedrine

Characters:

Suvai - karppu

Thanmai - veppam

Pirivu - karppu

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Action:

Stomachic, Carminative,Expectorant, Tonic.

14. SEERAGAM

Common name	Yóè ‹
Botanical name	Cuminum cyminum
Family	Apiaceae
Synonyms	Ü ¬ ê, YK, à ð ° ‹ ðdê ‹, «ð£ê ù ° « ì £K
Part used	Seed
Chemical constituents	Thymene,Cuminol,Cymene

Characters

Suvai - karppu

Thanmai - Thatpam

Pirivu - Inippu

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Action:

Carminative, stimulant, stomachic, Astringent.

ANNEXURE – II

BIO-CHEMICAL ANALYSIS OF KARISALANKANNI CHOORANAM

Preparation of the extract:

5gms of the trial drug was weighed accurately and placed in a 250ml clean beaker. Then 50ml of distilled water was added and dissolved well. Then it is boiled well for about 10 minutes. It is cooled and filtered in a 100ml volumetric flask and then it is make up to 100ml with distilled water. This fluid was taken for analysis.

QUALITATIVE ANALYSIS

S.NO	EXPERIMENT	OBSERVATION	INFERENCE
1.	**TEST FOR CALCIUM** 2ml of the above prepared extract is taken in a clean test tube. To this add 2ml of 4% Ammonium oxalate solution	A white precipitate is formed	Indicates the presence of calcium
2.	**TEST FOR SULPHATE** 2ml of the extract is added to 5% Barium chloride solution.	A white precipitate is formed	Indicates the presence of sulphate
3.	**TEST FOR CHLORIDE** The extract is treated with silver nitrate solution	No white precipitate is formed	Absence of chloride
4.	**TEST FOR CARBONATE** The substance is treated with concentrated Hcl.	No Brisk effervessence is formed	Absence of carbonate
5.	**TEST FOR STARCH** The extract is added with weak iodine solution	No Blue colour is formed	Absence of starch

6.	**TEST FOR FERRIC IRON** The extract is acidified with Glacial acetic acid and potassium ferro cyanide.	No blue colour is formed	Absence of Ferric iron
7.	**TEST OF FERROUS IRON** The extract is treated with concentrated Nitric acid and Ammonium thiocyanate solution	Blood red colour is formed	Indicates the Presence of ferrous Iron.
8.	**TEST FOR PHOSPHATE** The extract is treated with ammonium Molybdate and concentrated nitric acid	No Yellow precipitate is formed	Absence of Phosphate
9.	**TEST FOR ALBUMIN** The extract is treated with Esbach's reagent	No Yellow precipitate is formed	Absence of Albumin
10.	**TEST FOR TANNIC ACID** The extract is treated with ferric chloride.	Blue black precipitate is formed	Indicates the presence of Tannic acid
11.	**TEST FOR UNSATURATION** Potassium permanganate solution is added to the extract	It gets decolourised.	Indicates the presence of unsaturated compound
12.	**TEST FOR THE REDUCING SUGAR** 5ml of Benedict's qualitative solution is taken in a test tube and allowed to boil for 2 minutes and add 8-10 drops of the extract and again boil it for 2 minutes.	Colour develops	Indicates the presence of Reducing sugar
13.	**TEST FOR AMINO ACID** One or two drops of the extract is placed on a filter paper and dried well. After	Violet colour is formed	Indicates the presence of Amino acid

	drying, 1% Ninhydrin is sprayed over the same and dried well.		
14.	**TEST FOR ZINC** The extract is treated with Potassium Ferrocyanide.	No white precipitate is formed	Absence of Zinc.

Inference:

The extract prepared from the sample **Karisalankanni chooranam** contains calcium, sulphate, ferrous iron, tannic acid, reducing sugar and amino acid, unsaturated compound.

Biochemical Analysis report was given by **Mrs. N.Nagaprema, M.Sc., H.O.D, Bio Chemistry Department, Government Siddha Medical College, Palayamkottai.**

ANNUXURE III

ACUTE TOXICITY STUDY

Acute oral toxicity refers to those adverse effects occurring following oral administration of a single dose of a single dose of a substance or multiple doses given within 24 hours. Acute toxic class method (OECD guidelines 423, (2000) was followed to arrive at the maximum safety dose of the drug extracts. Three Wistar strain female albino rats (8-12 weeks old, 180-200g body weight) were used in each group. Single dose (2g/kg) of the *Karisalankanni chooranam* was orally administered to overnight fasted (food but not water withheld) animas while control animals received the vehicle (0.3%w/v CMC). Animals were observed individually after dosing at least once during the first 4 hrs and daily thereafter, for a total of 14days. Body weights of the animals were recorded. The other observations include changes for skin, fur, eyes and mucous membranes, respiratory, circulatory and autonomic and central nervous system and somatomotor activity and behavior pattern. At the end of 14 days, all animals were subjected to gross necropsy.

Statistics

Data are expressed as mean ± SEM; data analysed by one way ANOVA followed by Dunnet's multiple range tests to determine the significance of the difference between the control group and rats treated with test compounds.

* Values were considered significant at P < 0.5.

Results

Acute toxicity study

All of the rats fed with the food sample showed normal general behavior, respiratory pattern, cardiovascular signs, motor activities, reflexes and normal change in skin and fur.

S.No	Parameter	Control	Sample 2g/kg
1	White blood cells (xlOVul)	9.36±0.54	8.53±0.19
2	Hemoglobin (g/dl)	11.50tb0.26	12.09±1.42
3	Mean corpuscular volume	60.45±2.3	58.16±3.5
4	Mean corpuscular hemoglobin cone, (g/dl)	34.56±0.86	30.25±1.39
5	Platelet (xlO'/ul)	5.60±0.52	4.29±0.26
6	Red blood cell ($xl0^6$/ul)	3.87±0.24	1.29 ± 1.25

Hematological values of *Karisalankanni chooranam* in the acute toxicity study

Table - 2

S.No	Parameter	Control	Sample 2g/kg
1	White blood cells (xlOVul)	9.36±0.54	8.53±0.19
2	Hemoglobin (g/dl)	11.50tb0.26	12.09±1.42
3	Mean corpuscular volume	60.45±2.3	58.16±3.5
4	Mean corpuscular hemoglobin cone, (g/dl)	34.56±0.86	30.25±1.39
5	Platelet (xlO'/ul)	5.60±0.52	4.29±0.26
6	Red blood cell ($xl0^6$/ul)	3.87±0.24	1.29 ± 1.25

Values are expresses as Mean ± S.E.M.

All groups were treated with oral dose of 2g/kg body weight

No significant different from normal control

S. No	Parameter	Control	Sample 2g/kg
1	Glucose (mg/dl)	148.75±3.96	151.03 ±0.31
2	BUN(mg/dl)	34.26±1.23	30.18± 1.29
3	Creatinine(mg/ dl)	0.46±0.06	0.40±0.01
4	Total protein (g/dl)	5.48±0.23	6.91±0.18
5	Albumin (g/dl)	3.49±0.62	2.45 ±0.13
6	Total bilirubin (mg/dl)	0.26±0.02	0.31 ±0.13
7	AST (u/1)	141.5±3.76	138.23 ± 1.82
8	ALT (u/1)	86.36±1.75	83.28 ±1.48
9	ALP (u/1)	75.57±2.16	75.02 ± 3.62

Table - 3

S. No	Parameter	Control	Sample 2g/kg
1	Glucose (mg/dl)	148.75±3.96	151.03 ±0.31
2	BUN(mg/dl)	34.26±1.23	30.18± 1.29
3	Creatinine(mg/ dl)	0.46±0.06	0.40±0.01
4	Total protein (g/dl)	5.48±0.23	6.91±0.18
5	Albumin (g/dl)	3.49±0.62	2.45 ±0.13
6	Total bilirubin (mg/dl)	0.26±0.02	0.31 ±0.13
7	AST (u/1)	141.5±3.76	138.23 ± 1.82
8	ALT (u/1)	86.36±1.75	83.28 ±1.48
9	ALP (u/1)	75.57±2.16	75.02 ± 3.62

Blood chemical values of food sample in the acute toxicity study Values are expresses as Mean ± S.E.M.

All groups were treated with oral dose of 2g/kg body weight No significant different from normal control

Discussion and conclusion

In acute toxicity study for 14 days, at a dose of 2g/kg of *Karisalankanni chooranam* sample were chosen for the experiment. In the aspect of general behaviors, the rats treated with food sample at a single dose had no signs of behavior changes and toxic signs. The treated groups revealed no significant differences in body weight gain. The increase in body weight may have resulted from physiological changes in rats such as metabolism, food and water intake.

HAEMATINIC ACTIVITY

"Evaluation of Haematinic Activity of the *Karisalankanni chooranam* on Phenyl Hydrazine Induced Anaemic Rats"

MATERIALS AND METHODS

Phenyl hydrazine (PHZ) used for induction of Anaemia and the standard drug Haematenic - Vit B12 syrup was purchased from authorized suppliers.

Experimental Animals The experiment was conducted in Wistar Albino rats of either sex, weighed 150-200g from the animal house of Periyar college of pharmaceutical sciences, tiruchirapalli. The animals were maintained under standard laboratory condition with food and water *ad libitum.* The animals were allowed to acclimatize for 2 weeks before being subjected to experimental protocol. The animals were treated in line with the guide and care of laboratory animals as approved by the Institutional Animal Ethical Committee.

PROCEDURE

Induction of Anaemia The aim of the study is to evaluate the efficacy of *Karisalankanni chooranam* in iron deficiency anaemia. So anaemic wistar albino rats are used for study. Phenyl hydrazine an haemotoxicity chemical is used to induce anaemia in rats. Animals were divided into four groups containing six animals in each.

However, the result from animal health monitoring in the entire period of 14days showed no sign of morbidity and diseases.

The albino Wistar rats were healthy as shown by the normal appearance of general behavior, respiratory pattern, cardiovascular signs, motor activities, reflexes and normal change in skin fur.

With regards to hematological values, most of values in treated groups were normal in comparison with the control group. Significantly, some values were different from those of the control group such as RBC, MCV, MCHC, and platelet. However, such values are within the normal ranges. These variations may have resulted from variation among animal groups (Feldman et al., 2000) (Inala et al., 2002). Therefore, these results suggest that the test drug did not cause hematological or immunological defects in rats.

Furthermore, blood chemical examination was performed in order to evaluate any toxic effects on liver. In this study, the levels of these blood chemical values were minor changes and remained within the normal range (Casley and King, 1980) (Levine, 1995) (Angkhasirisap et al., 2002).

In conclusion, *Karisalankanni chooranam* sample given orally to Wistar rats did not produce toxicities.

Group I served as control and received regular rat food and drinking water *ad libitum.*

Group II, III and IV rats received phenylhydrazine 10 mg/kg for 8 days orally to induce anaemia in rats. Rats that developed anaemia with haemoglobin concentration lower than 13 g/dl were recruited for the study.

Tween 20 a vehicle received by group II.

Group III received standard haematinic drug vit B12 (10 ml/kg).

Group IV received *Karisalankanni chooranam* (400 mg/kg) diluted in a vehicle tween 20. **Treatment of the Animals** The rats were randomly divided into four groups (6 rats per group) and treated daily for 4 weeks. Group 2, 3 and 4 animals are anaemic induced by phenylhydrazine. Group I (control) - received only water and food. These animals are not induced by anaemia. Group II (negative control) - received only vehicle Tween 20 (10 ml/kg) Group III (positive control) - received only Vit B12 syrup (1 ml/rat) a standard drug. Group IV -received 400 mg/ kg of *Karisalankanni chooranam* respectively. All administrations were by oral intubation. The absolute dose of test drug given to the rat was calculated by the body surface area ratio between human intended dosages against rat. All the groups were treated orally as single dose daily for three week.

Haematological Parameters Analysis

All the rats were fasted overnight and 0.5 ml of blood was collected on next day by puncturing Retro orbital sinus using capillary tube. The blood was collected after induction of anaemia with PHZ and during the end of first, second and third weeks of treatments. The red blood cell count (RBC), haemoglobin concentration

(Hb), mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH) and Packed cell volume (PCV) were determined using Haematogical analyzer HA - 22.

Statistical Analysis All the results were expressed as mean ± SEM of six animals. Analysis of variance was performed by ONE WAY ANOVA followed by Dunnet's test. Probability values less than 0.01 were considered as significant. Result Oral administration of Phenylhydrazine for 8 days induced anaemia in rats. *Karisalankanni chooranam* used in the management of anaemia. As a result there is an increase in the haematological parameters within 3 weeks compared to other group of rats.

Haematinic activity of ***Karisalankanni chooranam*** in Phenyl hydrazine induced Anaemia in Rat Phenyl hydrazine caused significant decrease ($P < 0.05$) in Hb concentration, RBC count and haematocrit value in all rats indicating Anaemia. After the administration of test drug *Karisalankanni chooranam* , the hematological parameters are significantly increased ($P < 0.05$). The PHZ - induced Anaemia was significantly reversed within one week of treatment with the test drug, reaching maximum by the third week. The effect of Haematinic syrup was comparable to the test drug *Karisalankanni chooranam* .

DISCUSSION

Iron deficiency anaemia is a common disease affecting women especially in reproductive age. It is characterised by microcystic hypochromic RBC, in which MCV and MCH are reduced. It occurs due to defective haemoglobin synthesis. Iron in the body is used primarily for the synthesis of haemoglobin and normal erythropoiesis requires 20-25 mg of iron per day. Due to chronic blood loss, increased iron requirement, malabsorption of iron leads to iron deficiency. So administration of iron required to manage the disease without side effect. It can be prevented by increased dietary intake of iron and vit C for absorption. Administration of Phenylhydrazine a haemotoxicity chemical induce anaemia. Rats under the haemoglobin level 13 mg/kg are recruited for the study. The haematological parameters such as PCV, MCV, MCH and Hb are moniterd for three weeks. After three weeks of treatment with *Karisalankanni chooranam* the haematological parameters reach the normal levels.

Values are expressed in terms of mean ±SEM OF 6 rats(ANOVA)

Effect of phenylhydrazine (10 mg/kg, o.p daily for 8 days) on some haematological parameters (T=0) (RBC, Hb, PCV, MCH and MCV)

Parameters	**Group I (control)**	**GroupII (anaemic)**	**GroupIII (Standard)**	**GroupIV (test drug)**
Hb(g/dl)	19.4±1.22	15.2±1.04	18.2±0.56	17.5±0.22
Pcv(%)	59.45±1.10	50.12±2.10	60.22±1.16	55.48±0.96
RBC(xl06	7.24±0.89	5.84±0.23	6.02±0.24	6.76±0.26
MCV(fl)	76.88±0.12	99.48±0.46	93.72±0.48	79.18±0.36
MCH(pg)	24.24±0.18	20.24±4.12	29.27±0.88	25.34±0.22

Satistically significant $p<0.05$ (in comparison with negative control))

Haematological parameter of rat after one week treatment

Parameters	**Group I (control)**	**GroupII (anaemic)**	**GroupIII (Standard)**	**GroupIV (test drug)**
Hb(g/dl)	19.4±1.22	15.2±1.04	18.2±0.56	17.5±0.22
Pcv(%)	59.45±1.10	50.12±2.10	60.22±1.16	55.48±0.96
RBC(xl06	7.24±0.89	5.84±0.23	6.02±0.24	6.76±0.26
MCV(fl)	76.88±0.12	99.48±0.46	93.72±0.48	79.18±0.36
MCH(pg)	24.24±0.18	20.24±4.12	29.27±0.88	25.34±0.22

Values are expressed in terms of mean ±SEM OF 6 rats(ANOVA)

**P<0.05 as compared with negative control ***P<0.01 as compared with negative control

Haematological parameter of rats after two week treatment

Parameters	Groupl	Group II	GroupIII	GroupIV
Hb(g/dl)	19.6±0.12	16.4±1.23	19.2±1.45	19.4±0.36
PCV(%)	59.84±0.46	50.24±0.44	60.32±0.21	57.18±0.18
RBC(xl06/uI)	7.28±2.46	5.96±0.24	6.20±0.44	5.44±0.23
MCV(fl)	76.02±0.88	94.02±0.67	77.64±0.42	79.24±0.28
MCH(pg)	26.12±0.22	32.12±0.42	30.17±0.66	29.77±0.52

Values are expressed in terms of mean ±SEM OF 6 rats(ANOVA)

Statistically significant p<0.05 (in comparison with negative control)

Haematological parameter of rats after three week treatment

parameters	Groupl	GroupII	GroupIII	GroupIV
Hb(g/dl)	19.7±0.12	16.6±1.24	19.4±2.68	19.8±1.44
PCV(%)	60.02±0.12	50.64±1.65	60.40±0.22	59.26±1.36
RBC(xl06/ul)	7.32±0.22	6.02±1.64	7.02±0.21	7.16±1.86
MCV(fl)	77.24±1.22	80.01±0.16	76.62±0.89	75.78±2.67
MCH(pg)	25.01±0.11	25.12±2.16	27.24±0.24	24.86±2.26

**P<0.05 as compared with negative control ***P<0.01 as compared with negative control

Satistically significant p<0.05 (in comparison with negative control))

**P<0.05 as compared with negative control ***P<0.01 as compared with negative control

DISCUSSION

Iron deficiency anaemia is a common disease affecting women especially in reproductive age. It is characterised by microcystic hypochromic RBC, in which MCV and MCH are reduced. It occurs due to defective haemoglobin synthesis. Iron in the body is used primarily for the synthesis of haemoglobin and normal erythropoiesis requires 20-25 mg of iron per day. Due to chronic blood loss, increased iron requirement, malabsorption of iron leads to iron deficiency. So administration of iron required to manage the disease without side effect. It can be prevented by increased dietary intake of iron and vit C for absorption. Administration of Phenylhydrazine a haemotoxicity chemical induce anaemia. Rats under the haemoglobin level 13 mg/kg are recruited for the study. The haematological parameters such as PCV,MCV,MCH and Hb are moniterd for three weeks(15). After three weeks of treatment with Pitha Paandu Maathirai(PPM)the haematological parameters reach the normal levels.

CONCLUSION

In this study the oral administration of *Karisalankanni chooranam* significantly increases the haematological parameters from first week of treatement. This study concluded the haematinc activity of *Karisalankanni chooranam* was more effective than the standard haematinic drug.

I. CASE SUMMARY OF OUT-PATIENTS

S.No.	OP. No	Name	Age/ Sex	Occupation	Duration of the illness	Starting of Treatment	End of Treatment	No. of days Treated	Results
1.	12963	Kalaivani	24/F	Student	6 Months	08.02.2016	08.03.2016	30	Good
2.	12866	Janani	29/F	Computer operater	3 Months	08.02.2016	08.03.2016	30	Good
3.	13206	Sivaranjani	20/F	Student	6 Months	09.02.2016	09.03.2016	30	Good
4.	13962	Shanthi	46/F	Housewife	6 Months	11.02.2016	11.03.2016	30	Moderate
5.	13963	Pappathi	50/F	Coolie	6 Months	11.02.2016	11.03.2016	30	Good
6.	14074	Kalyani	57/F	Housewife	1 Months	11.02.2016	11.03.2016	30	Good
7.	15376	Rajeshwari	41/F	Housewife	3 Months	15.02.2016	15.03.2016	30	Good
8.	15622	Maheshwari	43/F	Housewife	1 Year	16.02.2016	16.03.2016	30	Good
9.	16301	Mary	52/F	Coolie	6 Months	18.02.2016	18.03.2016	30	Good
10.	17775	Geetha	40/F	Housewife	3 Months	22.02.2016	22.03.2016	30	Good
11.	18972	Murugan	42/M	Tailor	3 Months	25.02.2016	25.03.2016	30	Good
12.	19107	Anantham	60/F	Housewife	2 Months	26.02.2016	26.03.2016	30	Moderate
13.	19538	Chelladurachi	50/F	Coolie	1 Year	27.02.2016	27.03.2016	30	Good
14.	19597	Rekha	43/F	Computer Operator	1 Year	27.02.2016	27.03.2016	30	Fair
15.	20311	Gowdulaalam	60/M	Retired Teacher	6 Months	01.03.2016	30.03.2016	30	Good
16.	20371	Jesuraj	54/M	Loadman	6 Months	01.03.2016	30.03.2016	30	Moderate
17.	21436	Leela	57/F	Housewife	2 weeks	04.03.2016	02.04.2016	30	Good
18.	21913	Ganesan	45/M	Driver	1 Year	06.03.2016	04.04.2016	30	Good
19.	21914	Subramanium	50/M	Coolie	6 Months	06.03.2016	04.04.2016	30	Good
20.	24459	Sundari	33/F	Housewife	6 Months	15.03.2016	13.04.2016	30	Good

II. CASE SUMMARY OF IN-PATIENTS

S.No	IP. No	Name	Age / Sex	Occupation	Duration of the illness	Starting of Treatment	End of Treatment	No. of days Treated			Results
								IP	OP	Total	
1.	401	Pichammal	60/F	Housewife	3 Months	16.02.2016	16.03.2016	30	-	30	Good
2.	406	Jothi	23/F	Coolie	1 Year	16.02.2016	23.02.2016	08	22	30	Good
3.	478	Pappa	55/F	Coolie	1 Year	23.02.2016	23.04.2016	30	-	30	Good
4.	544	Gomathi	46/F	Housewife	6 Months	29.02.2016	29.03.2016	30	-	30	Good
5.	571	Shanmugakani	57/F	Housewife	1Year	02.03.2016	31.03.2016	30	-	30	Good
6.	611	Selvabackiyam	50/F	Coolie	6 Months	07.03.2016	01.04.2016	26	4	30	Good
7.	667	Subbu	55/F	Coolie	1 Year	10.03.2016	06.04.2016	28	2	30	Good
8.	736	Thaiaamal	57/F	Housewife	3 Months	18.03.2016	29.03.2016	12	18	30	Good
9.	953	Muthu	55/M	Coolie	6 Months	07.04.2016	25.04.2016	14	16	30	Good
10.	954	Ponnukutti	58/F	Housewife	1 Year	07.04.2016	20.04.2016	14	16	30	Moderate
11.	962	Sendu	60/F	Coolie	6 Months	08.04.2016	22.04.2016	15	15	30	Moderate
12.	964	Mookkammal	35/F	Housewife	3 Months	08.04.2016	18.04.2016	11	19	30	Good
13.	980	Pappa	55/F	Coolie	1 Year	09.04.2016	28.04.2016	20	10	30	Good
14.	983	Gurusamy	55/M	Coolie	6 Months	09.04.2016	08.05.2016	30	-	30	Good
15.	1000	Rathinammal	60/F	Housewife	1 Year	13.04.2016	12.04.2016	30	-	30	Fair
16.	1025	Varatharajan	50/M	Farmer	6 Months	16.04.2016	26.04.2016	11	19	30	Fair
17.	1070	Murugan	55/M	Coolie	6 Months	21.04.2016	17.05.2016	27	3	30	Good
18.	1105	Mohammed	52/M	Coolie	4 Months	25.04.2016	24.05.2016	30	-	30	Moderate
19.	1121	Subbammal	40/F	Coolie	6 Months	27.04.2016	26.05.2016	30	-	30	Good
20.	1148	Perumal	53/M	Coolie	1 Year	29.04.2016	28.05.2016	30	-	30	Good

III. LABORATORY INVESTIGATION REPORT OF THE OUT PATIENTS

S. no	OP No	Name	Age / Sex	Before treatment				After Treatment				ESR (mm)				Blood Sugar ®(gms/dl)		Urine analysis						Stools				
				TC (cu/mm	DC			TC (cu/mm	DC			BT		AT				BT			AT			BT		AT		
					P %	L %	E %		P %	L %	E %	½ hr	1 hr	½ hr	1 hr	BT	AT	Alb	Sug	Dep	Alb	Sug	Dep	Ova	Cyst	Ova	Cyst	OB
1	12963	Kalaivani	24 F	8000	66	31	3	9700	58	40	2	10	20	7	15	128	110	N	N	NAD	N	N	NAD	N	N	N	N	N
2	12866	Janani	29 F	8700	59	37	4	9000	60	36	4	10	20	5	10	83	80	N	N	NAD	N	N	NAD	N	N	N	N	N
3	13206	Sivaranjani	20 F	6100	64	34	2	7000	60	37	3	2	10	2	10	84	93	N	N	NAD	N	N	NAD	N	N	N	N	N
4	13962	Shanthi	46 F	6300	63	32	5	7300	60	36	4	7	25	7	15	99	110	N	N	NAD	N	N	NAD	N	N	N	N	N
5	13963	Pappathi	50 F	8000	60	40	5	8000	58	39	3	20	40	10	20	130	100	N	N	NAD	N	N	NAD	N	N	N	N	N
6	14074	Kalyani	57 F	9100	59	38	3	9800	57	40	3	15	47	7	35	83	85	N	N	NAD	N	N	NAD	N	N	N	N	N
7	15376	Rajeshwari	41 F	8700	59	39	2	8700	58	39	3	2	3	5	10	80	82	N	N	NAD	N	N	NAD	N	N	N	N	N
8	15622	Maheshwari	43 F	9000	56	40	4	9200	59	39	2	10	20	6	12	80	90	N	N	NAD	N	N	NAD	N	N	N	N	N
9	16301	Mary	52 F	8100	60	37	3	8700	59	38	3	10	22	5	15	135	120	N	N	NAD	N	N	NAD	N	N	N	N	N
10	17775	Geetha	40 F	8400	65	32	3	9000	60	37	3	3	12	5	10	100	88	N	N	NAD	N	N	NAD	N	N	N	N	N
11	18972	Murugan	42M	8800	63	35	2	9000	62	36	2	15	30	7	15	85	88	N	N	NAD	N	N	NAD	N	N	N	N	N
12	19107	Anantham	60 F	9500	80	18	2	9700	77	20	3	10	20	5	10	76	85	N	N	NAD	N	N	NAD	N	N	N	N	N
13	19538	Chelladurachi	50 F	7200	62	30	8	9100	60	38	2	10	20	6	12	115	120	N	N	NAD	N	N	NAD	N	N	N	N	N
14	19597	Rekha	43 F	9100	58	39	3	9100	60	38	2	15	30	10	20	110	90	N	N	NAD	N	N	NAD	N	N	N	N	N
15	20311	Gowdulaalam	60M	8000	58	36	6	8600	66	28	6	4	28	5	12	110	77	N	N	NAD	N	N	NAD	N	N	N	N	N
16	20371	Jesuraj	54M	8000	60	36	4	8600	61	36	3	7	15	6	12	90	80	N	N	NAD	N	N	NAD	N	N	N	N	N
17	21436	Leela	57 F	7500	66	30	4	8000	57	40	3	5	15	5	10	89	87	N	N	NAD	N	N	NAD	N	N	N	N	N
18	21913	Ganesan	45M	7500	57	39	4	8100	58	40	2	4	16	5	10	80	83	N	N	NAD	N	N	NAD	N	N	N	N	N
19	21914	Subramanium	50M	8100	54	43	3	8500	57	40	3	7	15	6	12	84	80	N	N	NAD	N	N	NAD	N	N	N	N	N
20	24459	Sundari	33 F	8100	60	36	4	8500	60	36	4	10	20	7	15	88	85	N	N	NAD	N	N	NAD	N	N	N	N	N

BT – Before Treatment, AT – After Treatment, N – Nil TC – Total Blood Count, DC – Differential Blood Count, P – Polymorphs, L – Leucocytes ,E-Eosinophils
ESR – Erythrocytes Sedimentation Rate, Alb – Albumin, Sug – Sugar, Dep– Deposits, OB – Occult blood

IV. LABORATORY INVESTIGATION REPORT OF THE IN PATIENTS

S. no	IP No	Name	Age / Sex	Before treatment				After Treatment				ESR (mm)				Blood Sugar ®(gms/dl)		Urine analysis						Stools				
				TC (cu/m	DC			TC (cu/mm	DC			BT		AT				BT			AT							
					P %	L %	E %		P %	L %	E %	½ hr	1 hr	½ hr	1 hr	BT	AT	Alb	Sug	Dep	Alb	Sug	Dep	Ova	Cyst	Ova	Cyst	OB
1	401	Pichammal	60/F	8500	58	39	3	9100	58	39	3	10	20	6	12	110	90	N	N	NAD	N	N	NAD	N	N	N	N	N
2	406	Jothi	23/F	9100	59	38	3	9600	58	40	2	10	20	5	10	90	88	N	N	NAD	N	N	NAD	N	N	N	N	N
3	478	Pappa	55/F	8400	57	39	4	9000	58	39	3	7	15	5	10	88	86	N	N	NAD	N	N	NAD	N	N	N	N	N
4	544	Gomathi	46/F	8100	60	37	3	8500	60	38	2	5	12	5	10	90	88	N	N	NAD	N	N	NAD	N	N	N	N	N
5	571	Shanmugakani	57/F	7800	60	36	4	8300	59	39	2	7	15	5	15	100	90	N	N	FPC	N	N	FPC	N	N	N	N	N
6	611	Selvabackiyam	50/F	8400	53	44	3	8800	53	45	2	5	12	5	10	90	88	N	N	NAD	N	N	NAD	N	N	N	N	N
7	667	Subbu	55/F	9000	60	38	2	9100	60	38	2	5	10	5	10	88	90	N	N	NAD	N	N	NAD	N	N	N	N	N
8	736	Thaiaamal	57/F	8300	59	38	3	8200	60	37	3	10	20	5	10	85	90	N	N	NAD	N	N	NAD	N	N	N	N	N
9	953	Muthu	55/M	8100	59	38	3	8500	59	39	2	7	15	5	15	83	80	N	N	NAD	N	N	NAD	N	N	N	N	N
10	954	Ponnukutti	58/F	8700	58	39	3	8800	59	39	2	10	20	5	10	85	88	N	N	NAD	N	N	NAD	N	N	N	N	N
11	962	Sendu	60/F	8500	57	40	3	8800	60	38	2	10	20	10	20	88	90	N	N	NAD	N	N	NAD	N	N	N	N	N
12	964	Mookkammal	35/F	7800	59	38	3	8100	59	38	3	5	10	5	10	88	85	N	N	NAD	N	N	NAD	N	N	N	N	N
13	980	Pappa	55/F	9600	59	38	3	9600	60	37	3	5	12	5	10	100	90	N	N	NAD	N	N	NAD	N	N	N	N	N
14	983	Gurusamy	55/M	7500	59	37	4	8600	59	39	2	2	5	5	10	83	80	N	N	NAD	N	N	NAD	N	N	N	N	N
15	1000	Rathinammal	60/F	7500	59	38	3	7500	59	38	3	10	22	7	15	90	85	N	N	NAD	N	N	NAD	N	N	N	N	N
16	1025	Varatharajan	50/M	8100	59	39	2	8300	59	39	2	5	10	7	15	85	87	N	N	NAD	N	N	NAD	N	N	N	N	N
17	1070	Murugan	55/M	7600	59	38	3	7700	59	38	3	6	20	5	10	88	86	N	N	NAD	N	N	NAD	N	N	N	N	N
18	1105	Mohammed	52/M	8000	60	38	2	8100	59	38	3	12	20	5	10	93	90	N	N	NAD	N	N	NAD	N	N	N	N	N
19	1121	Subbammal	40/F	9000	59	38	3	9100	60	37	3	10	20	7	15	90	88	N	N	FPC	N	N	FPC	N	N	N	N	N
20	1148	Perumal	53/M	7500	70	28	2	8100	60	37	3	20	45	10	20	86	88	N	N	NAD	N	N	NAD	N	N	N	N	N

BT – Before Treatment, AT – After Treatment, N – NilTC – Total Blood Count, DC – Differential Blood Count, P – Polymorphs, L – Leucocytes, E – Eosinophils
ESR – Erythrocytes Sedimentation Rate, mm – Millimeter , Sug-Sugar,Dep- Deposits, FPC – Few Pus cells, OB-Occult blood

BIOCHEMICAL AND HAEMATOLOGICAL REPORTS OF THE OUTPATIENT

S.No	OP No	Name	Age / Sex	Blood urea		Serum Creatinine		Hb(gms/dl)		RBC (millions/cu.mm)		PCV (%)		MCV (fl)		MCH (pg)		MCHC (%)		PBS	
				BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT
1	12963	Kalaivani	24 F	22	21	0.6	0.7	9.4	12.4	3.6	3.8	25	35	69	92	26.1	36.4	37.6	35.4	HM	HM
2	12866	Janani	29 F	26	23	0.6	0.6	8.3	11.5	3.8	4.0	26.7	34	74.1	89.4	23	30.2	31	33.8	HM	HM
3	13206	Sivaranjani	20 F	30	28	0.5	0.6	9.6	12.6	3.9	4.0	28	36	80	90	27.4	35	34.2	35	HM	NN
4	13962	Shanthi	46 F	32	30	0.8	0.8	9.2	11.5	3.8	3.9	26	33	76.4	91.6	27	31.9	35.3	34.8	HM	HM
5	13963	Pappathi	50 F	30	27	0.6	0.7	9.1	12.5	3.9	4.0	26.3	36	75.1	94.7	26	32.8	34.6	34.7	HM	NN
6	14074	Kalyani	57 F	28	28	0.6	0.7	8.8	12	3.9	4.0	26	36	70.2	92	23.7	46	33.8	33.4	HM	NN
7	15376	Rajeshwari	41 F	24	23	0.8	0.6	7.8	11.6	3.9	4.2	29	38	82.8	90	22.2	36.2	26.8	41.4	HM	NN
8	15622	Maheshwari	43 F	22	23	0.7	0.6	7.1	10.2	3.9	3.9	28	34	75.6	97	19.1	29	25.3	30	HM	HM
9	16301	Mary	52 F	23	24	0.9	0.8	7	10.5	3.8	4.10	29	37	82.8	87	20	29	24.1	28.4	HM	NN
10	17775	Geetha	40 F	24	23	0.6	0.6	7.7	10.4	3.9	4.1	30	38	85.7	95.5	22	28.8	25.7	27.4	HM	NN
11	18972	Murugan	42M	27	27	0.6	0.6	9.4	12.7	4.01	4.1	26.3	36	77.3	90	27.6	35.2	35.7	35.2	HM	NN
12	19107	Anantham	60 F	28	24	0.7	0.6	9.8	11.8	3.91	4.22	32.2	40	89.4	95.2	27	31	30.4	29.5	HM	HM
13	19538	Chelladurachi	50 F	27	25	0.8	0.8	9.9	12.4	4.0	4.1	29	35	80	92	30.9	36.4	34.1	35.4	HM	HM
14	19597	Rekha	43 F	35	30	0.7	0.6	7	8.2	3.9	4.0	25	28	73.5	77.8	20.5	22.7	28	29.2	HM	NN
15	20311	Gowdulaalam	60M	28	24	0.6	0.6	9.4	12.6	3.7	4.2	32	39	86.4	92.8	25.4	30	29.4	32.3	HM	NN
16	20371	Jesuraj	54M	22	23	0.6	0.6	9.8	11.5	3.5	3.8	30.2	29	82.8	97.3	28	30.2	33.7	31	HM	NN
17	21436	Leela	57 F	23	24	0.7	0.6	9.5	12.5	3.3	3.6	34.7	32	86.9	95.5	28.7	34.7	29.6	32.8	HM	NN
18	21913	Ganesan	45M	24	23	0.6	0.7	8.5	12	3.6	4.2	28.5	30	83.4	88.1	23.6	28.5	28.3	32.4	HM	NN
19	21914	Subramanium	50M	22	23	0.8	0.7	8	11.6	3.5	4.8	24.1	28	80	70.1	22.8	24.1	28.5	34.1	HM	NN
20	24459	Sundari	33 F	27	24	0.6	0.6	8.3	11.5	3.3	3.6	28	37	84.8	92.7	25.1	31.9	29.6	31	HM	NN

BT-BeforeTreatment , RBC-Red Blood Cells,PCV-Packed Cell Volume, MCV-Mean Corpuscular Volume, MCH-Mean Corpuscular Haemoglobin, MCHC-Mean Corpuscular Haemoglobin Concentration, PBS-Peripheral Blood Smear, HM-Hypochromic Microcytic, NN-Normochromic Normocytic

BIO CHEMICAL AND HAEMATOLOGICAL REPORTS OF THE IN PATIENT

S.No	OP No	Name	Age / Sex	Blood urea		Serum Creatinine		Hb(gms/dl)		RBC (millions/cu. mm)		PCV (%)		MCV (fl)		MCH (pg)		MCHC (%)		PBS	
				BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT
1	401	Pichammal	60/F	30	27	0.6	0.7	8	11.6	3.5	3.2	28	34	80	96.2	22.8	36.2	28.5	34.1	HM	HM
2	406	Jothi	23/F	28	27	0.6	0.6	9	12.1	3.4	3.3	31	38	81.1	85.1	26.4	36.7	29.1	31.8	HM	NN
3	478	Pappa	55/F	27	27	0.8	0.7	9.6	12	3.6	3.8	30	37	83.3	97.3	26.7	31.5	32	32.4	HM	NN
4	544	Gomathi	46/F	26	24	0.7	0.6	8.8	11.8	3.4	3.6	30	37	88.2	92.7	25.8	32.7	29.3	31.8	HM	NN
5	571	Shanmugakani	57/F	28	27	0.8	0.7	9.2	12.2	3.2	4.0	28	37	87.5	92.5	28.7	30.5	32.8	32.9	HM	NN
6	611	Selvabackiyam	50/F	27	23	0.6	0.6	9	12.5	3.7	3.5	32	38	86.4	98.5	24.3	35.7	28.1	32.8	HM	NN
7	667	Subbu	55/F	26	25	0.8	0.7	8.4	11.5	3.8	3.6	29	37	76.3	92.7	22.1	31.9	28.9	31.1	HM	NN
8	736	Thaiaamal	57/F	24	23	0.6	0.7	8.6	11.6	3.2	3.6	29	37	80.6	92.7	26.2	32.2	29.6	31.3	HM	NN
9	953	Muthu	55/M	24	23	0.6	0.8	9.8	12	3.5	3.8	31	38	88.5	96	28	31.5	31.6	31.5	HM	HM
10	954	Ponnukutti	58/F	26	23	0.6	0.6	8	10.2	2.8	3.4	28	35	80	92.9	28.5	30	28.5	29.1	HM	NN
11	962	Sendu	60/F	27	28	0.6	0.6	9.3	11.5	2.7	3.8	29	37	87.4	97.3	34.4	30.2	32.1	31.1	HM	HM
12	964	Mookkammal	35/F	27	24	0.6	0.7	8.4	11.5	3.8	4.2	28.3	37	74.4	88.1	22.1	27.3	29.6	31.1	HM	NN
13	980	Pappa	55/F	28	26	0.8	0.6	8.5	11.1	3.6	3.4	28	35	77.7	92.9	23.6	32.6	30.3	31.7	HM	NN
14	983	Gurusamy	55/M	21	23	0.6	0.6	8.8	11.8	3.5	4.2	30	38	85.7	90.4	25.1	28.1	29.3	31.1	HM	HM
15	1000	Rathinammal	60/F	27	25	0.8	0.7	9.2	10.5	3.6	3.8	31	38	86.1	96	25.6	27.6	29.6	27.6	HM	NN
16	1025	Varatharajan	50/M	23	22	0.7	0.6	7.6	8.7	3.4	4.2	27	30	79.4	71.4	22.3	20.7	28.1	29	HM	NN
17	1070	Murugan	55/M	24	22	0.6	0.6	8.5	11.5	3.2	4.2	29	37	90.6	88.1	26.5	27.3	29.3	31.1	HM	NN
18	1105	Mohammed	52/M	26	25	0.9	0.6	8.4	10.5	3.5	3.8	29	37	82.8	97.3	24	27.6	28.9	28.3	HM	HM
19	1121	Subbammal	40/F	28	24	0.8	0.8	7.2	10.5	3.6	3.8	26	35	72.2	92.1	20	27.6	27.6	30	HM	NN
20	1148	Perumal	53/M	28	27	0.7	0.6	9.1	12.5	3.8	4.2	28.3	37	90	98.3	22.1	27.3	28.4	32.1	HM	NN

BT-BeforeTreatment , AT-After Treatment ,RBC-Red Blood Cells,PCV-Packed Cell Volume, MCV-Mean Corpuscular Volume, MCH-Mean Corpuscular Haemoglobin, MCHC-Mean Corpuscular Haemoglobin Concentration, PBS-Peripheral Blood Smear, HM-Hypochromic Microcytic, NN-Normochromic Normocytic

DISCUSSION

Untreated Vatha pandu noi produces many complications.The symptoms of VathaPandu are closely related to Iron deficiency Anaemia.

Totally 40 patients were selected 20 patients were treated in OPD and 20 patients were treated in IPD of PG Pothumaruthuvam Department, Government Siddha medical College and Hospital, Palayamkottai. All the patients were administered with a trial drug **"Karisalankanni chooranam"**. The time duration of treatment was 30 days and all necessary investigations were carried out to all patients and followed up regularly in the OP & IP department.

Sex:

Among 40 cases, 29 Patients (72.5%) were females and 11 Patients(27.5%) were males.

Although Vathapandu affects both sexes. Females are mostly affected than males. This may be due to excessive menstrual blood loss which aggregates the already existing malnutrition.

Age:

Out of 40 patients, 21 Patients(52.5%) were in the age group of 51-60, 12 Patients(30%) were in the age group of 41-50, 3 Patients(7.5%) were in the age group of 31- 40, 3 Patients (7.5%)were in the age group of 21-30, 1 Patient (2.5%) were in the age group of 12-20.

Kaalam:

Out of 40 patients, 5 Patients(12.5%) comes under VathaKaalam and 35 Patients(87.5%) comes under PithaKaalam.

Paruvakaalam:

Out of 40 patients, 22 Patients(55%) comes under Pinpani, 11 patients (27.5%) come under Elavenil, and 7 Patients(17.5%) comes under Munpani kaalam.

.**Thinai:**

Out of 40 patients, 35 Patients(87.5%) comes under Marutham and 3 patients (7.5%) comes under Neithal, 2 patients(5%) comes under mullai nilam.

Occupation:

Out of 40 patients, 21 Patients(52.5%) were House wife, 12 Patients(30%) were Labours, 4 (10%)were office going and remaining 3 Patients(7.5%) are students.labours were more affected due to malnourished diet.

Socio-economical status:

Out of 40 patients, 33 Patients(82.5%) belongs to low income group , 7 Patients(17.5%) belongs to middle income group. Economically low income group were more affected than middle or high income group.

Diet:

Out of 40 patients, 31 Patients (77.5%) were mixed diet and 9 Patients (22.5%) were vegetarian.

Aetiological factors:

Out of 40 patients, 37 patients(92.5%) were due to nutritional deficiency,3 patients(7.5%) were due to blood loss. So it is evident that nutritional deficiency plays a major role in causing Iron deficiency Anaemia.

Mukkutram:

Vatham:

- Pranan was affected in all the patients 100% reflected as dyspnoea on exertion.
- Abanan was affected in 11 patients (27.5%) reflected as constipation and menorrhagia.
- Viyanan was affected in 35 patients (87.5%) reflected as headache and joint pain.
- Samanan was affected in all patients 100% reflected as loss of appetite.
- Kirugaran was affected in all patients 100% reflected as loss of appetite and Devathathan was affected in all patients 100% resulting in fatigue.

Pitham:

- Analagam was affected in all patients (100%) producing loss of appetite.
- Ranjagam was affected in all patients (100%) resulting in pallor of conjunctiva and nail bed.

- Sathagam was affected in all patients (100%) and Pirasagam was affected in 18 patients(45%) resulting in pallor of skin (Hb below 8gms/dl).

Kabham:

- Avalambagam was affected in all patients (100%) resulting in dyspnoea on exertion.
- Kilethagam was affected in all patients (100%) resulting in loss of appetite.
- Santhigam was affected in 14 cases (35%) resulting pain in knee joints.

Ezhu udal thathukkal:

- Saaram was affected in all patients (100%) causing tiredness.
- Senneer was affected in all patients (100%) producing pallor of conjunctiva and nail bed.
- Enbu was affected in 5 patients (12.5%) causing joint pain.

Ennvagai thervugal:

- All patients Naa, Niram and Vizhi were affected reflected as pallor of conjunctiva and nailbed.
- Malam was affected in 11 patients (27.5%) due to constipation.

Naadi:

- 30 of cases (75%) had Vatha Kapha Naadi, and 10 of cases (25%) had Kabha Pitha Naadi .

Neikuri:

- Out of 40 patients, 22 patients (55%) had Vatha Neer, 12 patients (30%) had Kabha Neer and 6 patients(15%) had Pitha Neer Neikuri.

Investigations:

TC, DC, ESR, Hb, PCV,MCV,MCH,MCHC,Peripheral blood smear, Blood sugar, Blood urea ,Serum creatinine and routine urine and motion test were taken and recorded before and after treatment.

Clinical Study:

All the patients were treated with **Karisalankanni chooranam** for an average of 30 days. Blood and urine was tested after the completion of treatment.

Clinical symptoms:

Out of 40 patients, 40 patients (100%) had Pallor of conjunctiva and nail bed, Fatigue, Loss of appetite, Dyspnoea on exertion, 29 Patients (72.5%) had Headache, 6 patients (15%) had Glossitis, 11patients (27.5%) had Constipation, 18 patients (45%) had Giddiness and Tachycardia.

Clinical Prognosis:

After 30 days of treatment Pallor of conjunctiva and nail bed ,Loss of appetite and Fatigueness present in 2 patients (5%), Dyspnoea on exertion present in 5 patients(12.5%),Headache present in 4 patients (10%), Glossitis present in 1 patient(2.5%).

Haemoglobin level:

After treatment 31 patients(77.5%) show increase in Hb 3gms/dl and above, 6 patients(15%) show increased in Hb 2 - 3gms/dl and 3 patients(7.5%) show increased in 1gm/dl.

Trial medicine:

- **Karisalankanni chooranam** was administered 2gm BD with sugar in hot water after food for 30 days.
- This drug stimulates appetite due to the presence of Chukku, Vitamin C is essential for iron absorption presence of Thiribala in the trial medicine it enhance the absorption.
- Usually oral iron therapy induces constipation, but this trial drug did not produce constipation as it contains thiribala which act as a laxative.

Grading of results:

Among 40 patients, 31 cases (77.5%) showed good result, 6 cases (15%) showed moderate result, 3 cases (7.5%) showed poor result.

SUMMARY

The **"Vathapandu Noi"**, a type of clinical condition with wide constitutional features was taken for this study.

The consummate literary collections from various siddhars has clearly mentioned the etiological factors, classifications of the disease and symptoms, complications,diet etc., according to their views.

The efficacy of the medicine **"Karisalankanni chooranam"** was studied and observed during this research work.

"Pandu" an ancient clinical entity with its historical importance correlated to the modern clinical entity **"Anemia"**. Anemia in every aspects of **"Vathapandu Noi"** with symptoms of pallor, anorexia, abdominal pain, indigestion, lasstitude, palpitation are noted with the clinical features discussed in modern medical text books.

The feasibility of considering **"Vatha pandu"** as **"Iron Deficiency Anemia"** is observed after considering the Siddha etiological factors which coincides to the modern aspects.

In this study 20 patients of both sexes of different age groups with classical clinical symptoms were selected as inpatients and another 20 patients were taken as out patients in post graduate department of Maruthuvam, Government Siddha Medical College and Hospital, Palayamkottai.

A total of 40 patients were treated in the OPD & IPD. The clinical and pathological assessment was carried out on the basis of both Siddha and modern aspects.

All the 40 patients were treated with Karisalankanni chooranam (2 gm/bd dailywith sugar in hotwater). The duration of the treatment was fixed as 30 days. The responses were assessed and recorded.

It'll be seen that well balanced diet is essential for overcoming this disease. Proper care food, manner and environment in which it is produced is of great importance in the preservation of good health.

Socio-economical status plays an important role in this aliment. The poor peoples are unhygeine with the usage of contaminated food and water, who were more prone to worm infestation.

The trial medicine is easily totarable by patients without any untoward effects and the medicine is very cheap.

Bio-chemical and pharmacological analysis reveal that the test medicine has significant haematinic effects.

CONCLUSION

The dissertation medicine “Karisalankanni chooranam” is the potent drug for the remedy of Iron Deficiency Anemia.

Research finding shows that 80% of the inpatients was improved well and 15% of the inpatients was moderately improved and 5% of the inpatients were poorly improved. 75 % of the outpatients was improved well and 15% of the outpatients was moderately improved and10 % of the outpatients was poorly improved.

The trial medicine was very effective to the patients and there was no recurrence of symptoms. The trial medicine is pure herbal formulation and it is free from side effects, so they are useful for long term purpose.

If the man strictly adheres to the Siddha’s principle he will lead a healthy and happy life.Out of 40 patients, 31 cases (77.5%) show good result, 6 cases (15%) show moderate result, 3 cases(7.5%) show mild result.

SUMMARY

The "**Vathapandu**" noi, a type of clinical condition with wide constitutional features was taken for this study.

The consummate literary collections from various siddhars has clearly mentioned the etiological factors, classifications of the disease and symptoms, complications,diet etc., according to their views.

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Socio-economical status plays an important role in this disease.

The trial medicine is easily tolarable by the patients without any side effects and the medicine is low cost.

Bio-chemical and pharmacological analysis revealed that the trial medicine has significant haematinic effects.

CONCLUSION

The open randamised clinical trial on medicine **"Karisalankanni chooranam"** is the highly potential drug for the remedy of Iron Deficiency Anemia.

Research finding showed that 80% of the inpatients was improved well and 15% of the inpatients was moderately improved and 5% of the inpatients were poorly improved. 75 % of the outpatients was improved well and 15% of the outpatients was moderately improved and10 % of the outpatients was poorly improved.

The trial medicine was very effective to Anaemia patients. The trial medicine is purely herbal formulation and it is free from side effects.

If the man strictly adheres to the Siddha's principle he will lead a healthy and happy life.

ANNUXURE IV

GOVERNMENT SIDDHA MEDICAL COLLEGE & HOSPITAL,

PALAYAMKOTTAI, TIRUNELVELI DISTRICT

DEPARTMENT OF POTHU MARUTHUVAM

PRECLINICAL AND PHASE II RANDOMIZED CLINICAL TRAIL ON

VATHA PAANDU (IRON DEFICIENCY ANAEMIA) WITH KARISALANKANNI CHOORANAM.

FORM-I

(SCREENING AND SELECTION PROFORMA)

1.Name_____ 2.Age_____ 3.gender_____ 4.Phone no _____

5. OP No. _____ 6. IP No. _____ 7. S.No. ._____

INCLUSION CRITERIA:

- Age :18- 60Yrs
- Sex : Both male and female
- Hb level between 7.1 – 10 grams%.
- Pallor of skin, mucous membrane, conjunctiva, nail beds.
- Fatigueness, shortness of breath, palpitation.
- Anorexia.
- Smear showing hypochromic microcytic.
- Worm infestation.
- Patients who are willing for admission and stay in IPD for 30days or willing to attend OPD
- Patients who are willing to give blood for laboratory investigation.
- Patient willing to sign the informed consent stating that he/she will consciously stick to the treatment during 30 days.

EXCLUSION CRITERIA

- Hb less than 7.

- Congenital heart disease.
- Patient with chronic renal disease.
- Liver disease.
- Inherited defects.
- Haemorrahagic disorders.
- Thalassemia.
- Ischaemic heart disease.
- Thyroid disorder.
- Diabetes mellitus.

DATE :

STATION : **SIGNATURE OF HOD**

SIGNATURE OF INVESTIGATOR **SIGNATURE OF LECTURER**

GOVERNMENT SIDDHA MEDICAL COLLEGE & HOSPITAL,

PALAYAMKOTTAI, TIRUNELVELI DISTRICT

DEPARTMENT OF POTHU MARUTHUVAM

PRECLINICAL AND PHASE II RANDOMIZED CLINICAL TRAIL ON

VATHA PAANDU(IRON DEFICIENCY ANAEMIA) WITH KARISALANKANNI CHOORANAM.

FORM l A

HISTORY PROFORMA ON ENROLLMENT

1. Serial No of the case: _____ 2. OP/IP No:_____

3. Name: _____ 4. Gender: Male ☐ Female ☐

5. Age (years): _____ DOB ☐☐ ☐☐ ☐☐☐☐
Date Month Year

6.Address: ----------

7.A.Occupation: ---------- B. Nature of work----------

8. Educational Status: A) Illiterate ☐ B)Literate ☐

9.Height:---------- cms 10.Weight:----------kg

11. Complaints and Duration:

_____

_____

_____

12. Past History

hypertension	_____
diabetes mellitus	_____
asthma	_____
pt	_____

HABITS

A) Smoking : 1. Yes ☐ duration _____ years; Number - _____ 2. No ☐

B) Alcoholism:1. Yes ☐ duration _____ years; Quantity- ____ ml 2. No ☐

C) Tobacco chewing: 1. Yes ☐ duration _____ years 2.No ☐

D) Betel chewing : 1. Yes ☐ duration _____ years 2.No ☐

13.Dietary style: A.Pure vegetarian ☐ B.Non-vegetarian ☐ C. Mixed diet ☐

14. Drug history: Had the patient been treated before with allopathy drug?

A) Yes ☐ 2) No ☐

15 Marital status **:** 1.Married ☐ 2.Unmarried ☐

16. Family history :

Whether this problem runs in family? 1. Yes ☐ 2.No ☐

If yes, mention the relationship of affected person(s) - _____

17. Menstrual history :

18. Bowel habits & micturition: Normal ☐

History of habitual constipation 1.Yes ☐ 2.No ☐

History of frequent diarrhoea 1.Yes ☐ 2.No ☐

History of frequent dysuria 1.Yes ☐ 2.No ☐

19. Psychological state: Normal ☐ Anxiety ☐ Depression ☐

Date :

Station :

Signature of the Investigator:

Signature of the Lecturer : **Signature of the HOD**

GOVERNMENT SIDDHA MEDICAL COLLEGE & HOSPITAL,

PALAYAMKOTTAI, TIRUNELVELI DISTRICT

DEPARTMENT OF POTHU MARUTHUVAM

PRECLINICAL AND PHASE II RANDOMIZED CLINICAL TRAIL

VATHA PAANDU(IRON DEFICIENCY ANAEMIA) WITH KARISALANKANNI CHOORANAM.

FORM II & II-A

CLINICAL ASSESSMENT ON ENROLLMENT AND ON VISITS

1. S.NO: _____ 2. OP/IP NO : _____

3. Name : _____ 4. Gender : _____

5. Date of assessment : _____

SIDDHA SYSTEM OF EXAMINATION

1**. ENVAGAI THERVU: [EIGHT-FOLD EXAMINATION]**

I. NAADI: [PULSE PERCEPTION]

	0^{st}Day	07^{th}Day	15^{th} Day	21^{st} Day	28^{th} Day	30^{th} Day
Vali						
Azhal						
Iyyam						
Vali Azhal						
Azhal vali						
Iyya vali						
Vali Iyyam						

Azhal Iyyam						
Iyya Azhal						

II. NAA:[TONGUE]

	0th Day	**07th Day**	**14th Day**	**21st Day**	**28th Day**	**30th Day**
Colour	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale
Taste	Sweet/ Bitter/ Sour/ Pungent/ None	Sweet/ Bitter/ Sour/ Pungent/ None	Sweet/ Bitter/ Sour/ Pungent/ None	Sweet/ Bitter/ Sour/ Pungent/ None	Sweet/ Bitter/ Sour/ Pungent/ None	Sweet/ Bitter/ Sour/ Pungent/ None
Coating	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent
Fissure	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent
Saliva	Normal/ Increased/ Decreased	Normal/ Increased/ Decreased	Normal/ Increased/ Decreased	Normal/ Increased/ Decreased	Normal/ Increased/ Decreased	Normal/ Increased/ Decreased
Dryness	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent
Glossitis	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent
Baldness	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent

III.NIRAM: [COMPLEXION]

0^{th} Day	07th day	14th Day	21st Day	28th Day	30th day
Dark/ Yellow/ tinted/ Pale	Dark/ Yellow/ tinted/ Pale	Dark/ Yellow/ tinted/ Pale	Dark/ Yellow/ tinted / Pale	Dark/ Yellow/ tinted/ Pale	Dark/ Yellow/ tinted/ Pale

IV.MOZHI: [VOICE]

0^{th} Day	07th day	14th Day	21st Day	28th Day	30th day
Medium/ High/ Low / Pitched	Medium/ High/ Low/ Pitched	Medium/ High/ Low/ pitched	Medium/ High/ Low/ pitched	Medium/ High/ Low/ pitched	Medium/ High/ Low/ pitched

V.VIZHI: [EYES] (Lower palpabrel conjunctiva)

0^{th} Day	07th day	14th Day	21st Day	28th Day	30th day
Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale

VI. MALAM; [BOWEL HABITS / STOOLS]

	0^{th} Day	07th Day	14th Day	21stDay	28th Day	30th day
Colour	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale	Dark/ Yellow/ Red/ Pale
Consistency	Solid/ Semisol	Solid/ Semisoli	Solid/ Semisoli	Solid/ Semisoli	Solid/ Semisoli	Solid/ Semisoli

	id/ Watery	d/ Watery	d/ Watery	d/ Watery	d/ Watery	d/ Watery
Stool bulk	Normal / Reduced	Normal/ Reduced	Normal/ Reduced	Normal/ Reduced	Normal/ Reduced	Normal/ Reduced
Constipation	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent
Diarrhoea	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent

VII. URINE EXAMINATION:

NEERKURI	**0th Day**	**07th day**	**14th Day**	**21st Day**	**28th Day**	**30th Day**
Niram [Colour]	White/ Yellowish/ Straw coloured / Crystal clear	White/ Yellowish/Straw Coloured / Crystal clear	White/ Yellowish/ Straw coloured/ Crystal clear	White/ Yellowish/ Straw coloured/ Crystal clear	White/ Yellowish/ Straw coloured/ Crystal clear	White/ Yellowish / Straw coloured/ Crystal clear
Manam [Odour]	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent
Nurai [Froth]	Nil/ Reduced / Increased	Nil/ Reduced/ Increased	Nil/ Reduced/ Increased	Nil/ Reduced/ Increased	Nil/ Reduced/ Increased	Nil/ Reduced/ Increased
Edai [Sp.gravity]	Normal/ Increased/	Normal/ Increased /Reduced	Normal/ Increased /Reduced	Normal/ Increased /	Normal/ Increased /	Normal/ Increased/ Reduced

	Reduced			Reduced	Reduced	
Enjal [Deposits]	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent	Present/ Absent
Volume	Normal/ Increased/ Reduced	Normal/ Increased /Reduced	Normal/ Increased /Reduced	Normal/ Increased / Reduced	Normal/ Increased / Reduced	Normal/ Increased/ Reduced

NEIKURI	**0th Day**	**7th day**	**14th Day**	**21st Day**	**28th Day**	**30th Day**
Serpentine fashion						
Annular/Ringed fashion						
Pearl beaded fashion						
Mixed fashion						
Other fashion						

VIII. SPARISAM: [PALPATORY PERCEPTION]

0th Day	**7th day**	**14th Day**	**21st Day**	**28thDay**	**30th day**
Warmth/ Cold/ Sweat	Warmth/ Cold/ Sweat	Warmth/ Cold/ Sweat	Warmth/ Cold/ Sweat	Warmth/ Cold/ Sweat	Warmth/ Cold/ Sweat

5. THEGI: [TYPE OF BODY CONSTITUTION]

Vatham predominant		Kabam predominant	
Pitham predominant		Thondha udal	

6.NILAM: [LAND WHERE PATIENT LIVED MOST]

Kurinji ☐ Mullai ☐ Marutham ☐ Neithal ☐ Palai ☐

(Hilly terrain) (Forest range) (Plains) (Coastal belt) (Arid regions)

7. KAALAM:

Kaarkalam - ☐ Pinpanikalam - ☐

Koothirkalam - ☐ Ilavenil - ☐

Munpanikalam - ☐ Muthuvenil - ☐

8. GUNAM:

Sathuvam - ☐ Rasatham - ☐ Thamasam - ☐

9.IMPORIGAL (SENSORY ORGANS):

	0th Day	**7th day**	**14th Day**	**21st Day**	**28th Day**	**30th Day**
Mei (Skin)						
Vai (Buccal Cavity)						
Kann (Eye)						
Sevi (Ear)						
Mooku (Nose)						

10. KANMENDRIYAM (MOTOR ORGANS)

	0th Day	**7th day**	**14th Day**	**21st Day**	**28th Day**	**30th Day**
Kai (upper limb)						
Kaal (lower limbs)						
Vai (buccal cavity)						
Eruvaai (excretory organs)						
Karuvaai (reproductive organs)						

11.KOSANGAL(Sheath)

	0th Day	**7th day**	**14th Day**	**21st Day**	**28th Day**	**30th Day**
Annamaya Kosam						
Pranamaya kosam						
Manomaya kosam						
Vignanamaya kosam						
Ananthamaya kosam						

12. MUKKUTRAM:[AFFECTION OF THREE HUMORS]

A)VATHAM:

	0th Day	**7th day**	**14th Day**	**21st Day**	**28th Day**	**30th Day**
Praanan						
Abaanan						
Viyaanan						
Udhaanan						
Samanan						
Naagan						
Koorman						
Kirukaran						
Devatha-than						
Dhanan-jeyan						

B) PITHAM:

	0th Day	**7th day**	**14th Day**	**21st Day**	**28th Day**	**30th Day**
Analpitham						
Ranjagam						
Saathagam						
Praasagam						
Aalosagam						

C) KABHAM:

	0th Day	**7th day**	**14th Day**	**21st Day**	**28th Day**	**30th Day**
Avalambagam						
Kilaethagam						
Pothagam						
Tharpagam						
Santhigam						

1.SEVEN UDALTHATHUS: (7 SOMATIC COMPONENTS)

	0th Day	**7th day**	**14th Day**	**21st Day**	**28th Day**	**30th Day**
Saaram [Chyme]						
Senneer [Blood]						
Oon [Muscle]						
Kozhuppu [Fat]						
Enbu [Bones]						
Moolai [Bone marrow]						
Sukkilam or suronitham [Genital discharges]						

14. SYSTEMIC EXAMINATION:

	0th Day	**7th day**	**14th Day**	**21st Day**	**28th Day**	**30th Day**
Locomotor system						
Cardiovascular system						
Respiratory system						
Gastro intestinal system						
Central nervous system						
Urogenital system						
Endocrine system						

15. GENERAL EXAMINATION:

	0th Day	**7th Day**	**14th Day**	**21st Day**	**28th Day**	**30th Day**
Height (cms)						
Weight (kg)						
Temperature (F^0)						
Pulse rate (per min)						
Heart rate (per min)						
Respiratory rate (per min)						

Blood pressure (mm/Hg)						
Pallor						
Jaundice						
Cyanosis						
Lymphadenopathy						
Pedal edema						
Clubbing						
Jugular vein pulsation						

Signs and symptoms:

S.NO	**Assesment**	0 th day	7 th day	14 th day	21 st day	28 th day	30 th day
1.	Pallor of conjunctiva and nail bed						
2.	Fatigue						
3.	Loss of appetite						
4.	Dyspnoea on exertion						
5.	Headache						
6.	Constipation						
7.	Glossitis						
8.	Giddiness						
9.	Tachycardia						

Date :

Station :

Signature of the Investigator:

Signature of the Lecturer : **Signature of the HOD**

GOVERNMENT SIDDHA MEDICAL COLLEGE AND HOSPITAL
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POST-GRADUATE DEPARTMENT OF POTHU MARUTHUVAM

PRECLINICAL AND PHASE II RANDOMIZED CLINICAL TRAIL ON

VATHA PAANDU(IRON DEFICIENCY ANAEMIA) WITH KARISALANKANNI CHOORANAM.

FORM III-LABORATORY INVESTIGATIONS

1	Sl.No		2	OP/IP No	
3	Bed No		4	Name	
5	Age		6	Gender	

I.BLOOD

		Before Treatment	After Treatment
1	TC (cells/mm)		
2	DC (%)		
	a)Neutrophils		
	b)Lymphocytes		
	cMmonocytes		
	d)Eosinophils		
3	ESR(mm)		
	a)1/2 hour		
	b)1 hour		
4	Haemoglobin		
5	Blood sugar		
6	Blood urea		
7	Serum creatinine		

8	Serum cholesterol		
9	PCV		
10	MCV		
11	MCH		
12	MCHC		
13	TOTAL RBC		
14	Platelet count		
15	Total iron binding capacity		

II.URINE

		Before Treatment	After Treatment
1	Albumin		
2	Sugar		
3	Epithelial cells		
4	Pus cells		
5	Red blood cells		
6	Casts/Crystals		

Date :

Station :

Signature of the Investigator:

Signature of the Lecturer : **Signature of the HOD**

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PRECLINICAL AND PHASE II RANDOMIZED CLINICAL TRAIL ON VATHA PAANDU (IRON DEFICIENCY ANAEMIA) WITH KARISALANKANNI CHOORANAM.

CONSENT FORM-IV A

Certificate by Investigator

I certify that I have disclosed all details about the study in the terms readily understood by the patient.

Date: …….. Signature: ……

Name: ……..

Consent by Patient

I have been informed to my satisfaction, by the attending physician, the purpose of the clinical trial, and the nature of drug treatment and follow-up including the laboratory investigations to be performed to monitor and safeguard my body functions.

I am aware of my right to opt out of the trial at any time during the course of the trial without having to give the reasons for doing so.

I, exercising my free power of choice, hereby give my consent to be included

As a subject in the clinical trial of **KARISALANKANNI CHOORANAM for the management of VATHA PAANDU (IRON DEFICIENCY ANAEMIA).**

Date: …….. Signature: ……

Name: ……..

Date: ……. Signature of Witness: ……..

Name…….. ……

Relationship:……

murdh rjj kUjJtf fyY}h kwWk kUjJtkid›
ghisaqNfhlil›
gllNkwgbgG nghJ kUjJtjJiw.
thjghzL NehafF kUejhf fhryhqfz z #uzk
ghfhgGjjwidf fzlwAk kUjJt MaT.

xgGjy gbtk

Mathsuhy rhdwsffgglIJ

ehd ,ej MaT Fwjj midjJ tguqfisAk NehahsfF GhAk tifay vLjJiujNjd vd cWjasffNwd.

Njj : ifnahggk :

,lk : ngah :

Nehahsad xgGjy

vddlk ,ej kUjJt Matd fhuzjijAk kUejd jdik kwWk kUjJt toKiwiag gwwAk njhlheJ vdJ cly ,affjij fzfhzffTk› mjidg ghJfhffTk gadgLk kUjJt MaTf$l ghNrhjidfs gwwjUgj msfFk tifay MaT kUjJtuhy tsfff $wgglIJ.

ehd ,ej kUjJt Matd NghJ fhuzk vJTk $whky vgnghOJ NtzLkhdhYk ,ej matyUeJ vdid tLtjJf nfhsSk chikia njhejUffNwd.

ehd vdDila Rjejukhf NjhT nraAk chikiaf nfhzL ‘thjghzL NehafF fhryhqfz z #uzk” ghfhgGjjwidf fzlwAk kUjJt MatwF vdid clgLjj xgGjy msffNwd.

Njj : ifnahggk:
,lk : ngah :

Njj: rhlrffhuh ifnahggk :
,lk: ngah:
cwTKiw :

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PRECLINICAL AND PHASE II RANDOMIZED CLINICAL TRAIL ON

VATHA PAANDU(IRON DEFICIENCY ANAEMIA) WITH KARISALANKANNI CHOORANAM.

FORM IV B

WITHDRAWAL FORM

Name: _____ OPD/ IPD number: _____

Age : _____

Date of trial commencement: _____

Date of withdrawal from trial: _____

Reasons for withdrawal:

- Long absence at reporting : Yes ☐ No ☐
- Irregular treatment : Yes ☐ No ☐
- Shift of locality : Yes ☐ No ☐
- Increase in severity of symptoms : Yes ☐ No ☐
- Development of severe adverse drug reactions : Yes ☐ No ☐

Date :

Station :

SIGNATURE OF INVESTIGATOR **SIGNATURE OF HOD**

SIGNATURE OF LECTURER

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DEPARTMENT OF MARUTHUVAM

PRECLINICAL AND PHASE-II RANDOMIZED OPEN CLINICAL STUDY ON VATHA PAANDU(IRON DEFICIENCY ANAEMIA) WITH KARISALANKANNI CHOORANAM.

FORM IV C
PATIENT INFORMATION SHEET

- This disease is not contagious.
- It is a clinical syndrome occurs due to deficiency of Iron.
- Many herbal and mineral siddha medicines are currently practiced by the siddha practioners for Anaemia.
- The trial drug is prescribed only with evidence of siddha literature.
- The trial drug is prepared at the Gunapadam lab of government siddha medical college & hospital, palayamkottai. under the direct supervision of teaching faculties of Maruthuvam and Gunapadam Dept.

Details of the trial drug:

1. **KARISALANKANNI CHOORANAM:**

DOSAGE : Two grams, two times after food
ADJUVANT : Sugar
Duration : 30 days .

- ❖ Patients are advised to take green vegetables, greens , protein foods, fibre foods, iron rich foods like dates, fig.
- ❖ Patients must walk 30-45 minutes per day
- ❖ Patients are advised to avoid tamarind, betel chewing, tobacco , alcohol and smoking.

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PALAYAMKOTTAI, TIRUNELVELI DISTRICT

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PRECLINICAL AND PHASE II RANDOMIZED CLINICAL TRAIL ON

VATHA PAAND(IRON DEFICIENCY ANAEMIA) WITH KARISALANKANNI CHOORANAM.

FORM IV D

ADVERSE DRUG REACTION FORM

Name: _____ OPD/ IPD No : _____

Age: _____

Date of trial commencement: _____

Date of withdrawal from trial: _____

Description of adverse reaction: _____

_____

Date:

Station:

SIGNATURE OF INVESTIGATOR **SIGNATURE OF HOD**

SIGNATURE OF LECTURER

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PRECLINICAL AND PHASE II RANDOMIZED CLINICAL TRAIL ON

VATHA PAANDU(IRON DEFICIENCY ANAEMIA) WITH KARISALANKANNI CHOORANAM.

FORM IV –E

(DRUG COMPLIANCE FORM)

Name : _____ Age/ Sex : _____ S. No : _____

OPD/ IPD No : _____ Date : _____ Bed No : _____

Name Of The Drug : **KARISALANKANNI CHOORANAM**

Drugs issued date :

Drugs returned date :

S.NO	DATE	DRUG TAKEN TIME	
		MORNING TIME	EVENING TIME
Day 1			
Day 2			
Day 3			
Day 4			
Day 5			
Day 6			
Day 7			
Day 8			
Day 9			

S.NO	DATE	MORNING/TIME	EVENING/TIME
Day 10			
Day 11			
Day 12			
Day 13			
Day 14			
Day 15			
Day 16			
Day 17			
Day 18			
Day 19			
Day 20			
Day 21			
Day 22			
Day 23			
Day 24			
Day 25			
Day 26			
Day 27			
Day 28			
Day 29			
Day 30			

Date :

Station :

SIGNATURE OF INVESTIGATOR **SIGNATURE OF HOD**

SIGNATURE OF LECTURER

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